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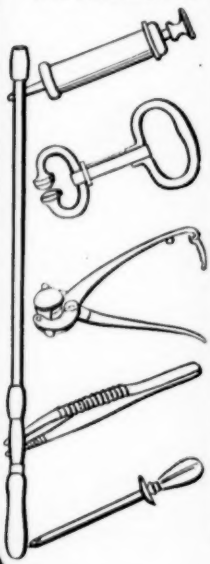

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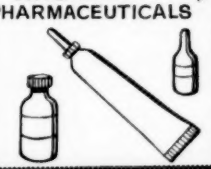

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Trends in Small-animal Practice

THE publication, in this issue, of articles on two subjects of considerable everyday importance to those engaged in small-animal practice, makes a review of the present situation in this sphere of veterinary activity opportune.

Despite the problems referred to in Dr. Howell's article—problems which it behoves everyone connected with dog practice either directly or *via* research to keep actively in mind and constantly to investigate and evaluate—the situation regarding vaccination in the canine species can be regarded as reasonably satisfactory. Living virus vaccines of the canine distemper group are safe, and confer an adequate and long-lasting immunity in a high proportion of cases provided they are used with care and thought by those injecting them. But whilst the increasing competition in the production of this type of vaccine should ensure the maintenance of a high standard in canine biologicals, it is none the less vital that veterinary surgeons should make their choice of material on sound clinical and scientific evidence unfettered by commercial inducement. This jealously-guarded freedom of action and choice is comparatively easy to maintain in pet practice where economic considerations can, and should, be subordinate to safety and efficiency.

There is, increasingly, a welcome awareness among dog owners of the value of vaccination, and this is particularly noticeable in dog breeding establishments and boarding kennels, where, in the bad old days, so many disastrous epidemics had their origin. To-day, in many parts of the country, serious epidemics of the canine distemper complex are becoming comparatively rare, although pockets of infection persist in homes for stray dogs and in the less scrupulous boarding and dealing establishments.

This encouraging situation, however, must not create complacency. It is essential that veterinary surgeons and owners alike should never relax the vaccination programme so that the dangerous situation of a highly susceptible dog population could recur.

Disease of the canine external ear remains a constant and, as yet, largely unsolved problem. Despite the excellent work of Dr. Fraser and others, much fundamental work remains to be done before great advances in this field may be expected. Fluctuations in breed popularity account for variations in the types of ear disease encountered, but until far more is known of the histology of normal and diseased ears in the various breeds of dog, the practising veterinary surgeon will be faced with this problem daily.

Another field where there can be no room for complacency is that of diseases, hormonal and otherwise, of the female reproductive system in the dog. Pyometra, severe pseudo-pregnancies and various forms of dystokia, particularly that due to uterine inertia, are all far too frequent for anyone, dog breeder or veterinary surgeon, to feel happy about the present situation. Few doubt that there is at least a familial predisposition in these cases perhaps less obvious, but as perilous, as frank congenital abnormalities which are causing so much anxiety in some fashionable breeds to-day.

Whilst veterinary surgeons have an undoubted duty to alleviate the crippling effects of such congenital defects as luxation of the patella and malposition of the tibial crest in any individual patient, they have an equally obvious—and far more constructive—duty to preach the policy of breeding only from sound stock. In some breeds the situation is so serious that the more far-sighted fanciers are themselves starting a crusade to eradicate these defects from their breeds.

In the surgical field steady progress continues. Anaesthetic methods and orthopaedic techniques continue to improve, and the good work started largely by those having access to every facility, as in the veterinary schools, is being increasingly known to those in average private practice. Thus to-day's patient in need of surgical assistance is likely to benefit from a high standard of anaesthetic technique and operative treatment in almost any practice where it may be presented.

Lest it be felt that we are concentrating too much on the dog, let it be added that the cat receives an ever increasing service from the profession. It is particularly pleasing to find so many cat owners desirous of availing themselves of the advances which veterinary medical and surgical practice have to offer every species of small-animal patient.

The position of practice in so-called exotic pets can be viewed with modest satisfaction. Veterinary surgeons have been quick to realise the need for attention to any species of household pet they may be asked to help, and a number are giving up much

(*Concluded at foot of page overleaf*)

Vaccination of the Dog*

BY

D. G. HOWELL

Glaxo Laboratories Ltd., Greenford

DURING the course of this paper and the ensuing discussion it will become increasingly clear that there are many questions on this subject to which the answers are unknown. The purpose of my paper will be to focus attention on some of these; if it seems that here and there I am being somewhat dogmatic, I must deny any intention of being so, my intention being rather to provoke a lively discussion. I am delighted to see present so many of my practising colleagues and of those interested in canine virus research.

Within the past 2 or 3 years veterinary medicine has seen the introduction of several different canine vaccines. It is timely therefore that we should take stock of the situation and ask one or two questions on the position of the average practitioner, and indeed of the research worker, which is bewildering, to say the least. Unfortunately veterinary medicine —particularly small-animal practice—is bedevilled by considerations of a commercial and economic nature resulting in the adoption of procedures that would be unacceptable in other spheres of medicine. Further, there is a distinct tendency to give the client what he wants, rather than what is necessarily best: in consequence the advertising acumen of the different pharmaceutical houses is undoubtedly having a marked effect on the way practice is conducted. In making statements of this kind I find myself a kind of Jekyll and Hyde; though we would all like to be Jekyll, Hyde appears to be gaining the upper hand. I am sure many colleagues find themselves in the same position.

There are 4 aetiological agents with which we are concerned—the distemper and hepatitis viruses and two species of Leptospira that parasitise the dog.

Distemper

I make no excuse for including hard pad under this heading. I am quite unrepentant as to the line I took at the 1959 B.S.A.V.A. Congress and would like to see this most unsatisfactory descriptive term abolished. If we wish to define in clinical terms different manifestations of distemper, I would prefer us to talk either of the catarrhal or the encephalitic

*A paper read to Central Veterinary Society, March 3rd, 1960.

Trends in Small-animal Practice.—*Concluded.*

time to disseminating information in this field through papers read to meetings, assisting in the preparation of handbooks and so on. One hopes that future generations, who come to profit from this work and who will be accepting exotic pet practice—perhaps no longer called exotic!—as an everyday matter, will realise that the profession was very ready to move with the times and meet the demand for this type of service.

That small-animal practitioners are very much alive to what might be called public health obligations was well seen at a recent meeting of the Central Veterinary Society, when members urged energetic action following warnings from two speakers of the possible danger to human health from certain types of monkey which are very liable to harbour B virus.

In this connexion it is a pity that the few members of the medical profession who have strong views on domestic pets as sources of infection to man, and who are sometimes responsible for what may be called unnecessarily alarmist reports in the Press, are not better aware that veterinary surgeons will always be alive to human risk whenever they diagnose a disease communicable from animal to man and will take all steps to safeguard human contacts.

Tuberculosis in domestic pets, especially in the cat, is undoubtedly decreasing with the eradication of bovine tuberculosis from this country; but even so occasional cases still occur and keep us on our guard. The control of human tuberculosis by modern chemo-therapy and antibiotics has decreased the risk of canine infection, which the profession has always realised was often passed from man to dog.

Against this, two conditions are receiving increasing attention, although it is as yet too early to assess their true significance in relation to human health. We refer to the fact that the common roundworms of the dog and cat have now been incriminated in cases of visceral larva migrans in children, and that *Toxoplasma gondii* is a protozoon which can be responsible for still births and mental defects in babies. As this latter organism is almost universally parasitic in the United Kingdom it seems rather hard that the household dog should be quite so vigorously blamed as it was in a recent article; yet it is a point to watch.

Let there be no doubts, however, that veterinary surgeons are fully aware of their obligations in these respects, while at the same time resisting any exaggerated suggestions that domestic pets as usually kept are a serious hazard to human health.

Progress in small-animal work continues at a steady pace, but, as in every other aspect of our work, for every problem apparently solved, several new ones appear to maintain the challenge necessary to any progressive scientific profession.

D. G. HOWELL.—VACCINATION OF THE DOG

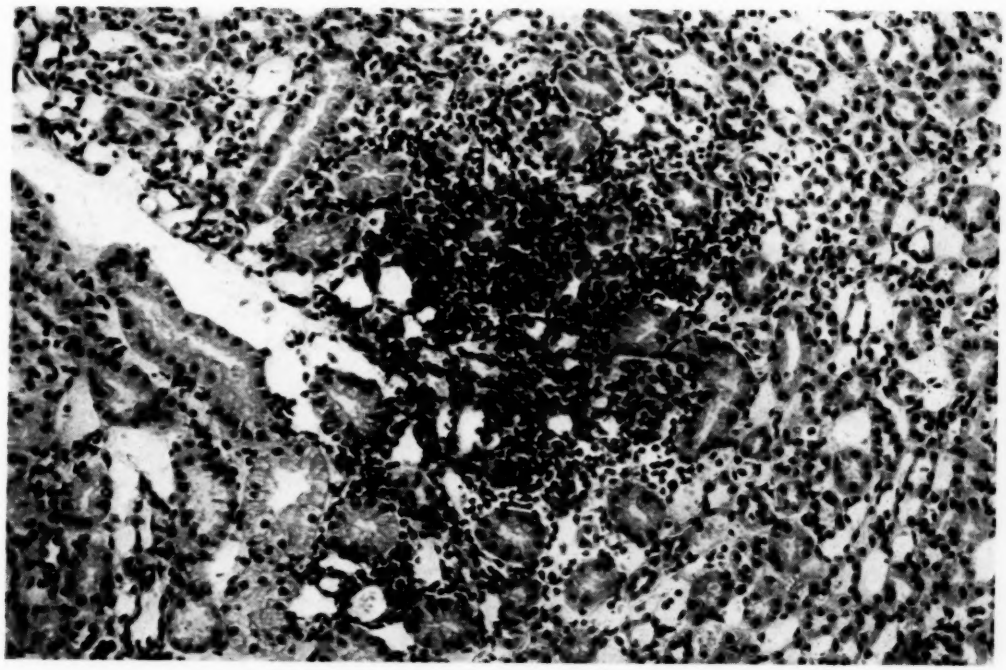


FIG. 1. Focal interstitial nephritis observed in dog No. 2 (Table II) examined 28 days after vaccination (Stained H. and E. x 200)

form and to recognise degrees of difference. The public appear to think, and with every justification, that hard pad disease is an entity distinct from distemper and that the present avianised distemper vaccines immunise against 2 diseases and by inference against 2 distinct viruses. No vaccine, either human or veterinary, has been developed to do this in a practical manner, and I am sure that the 2 classical strains of egg-adapted distemper virus independently developed in South Africa and the United States are not in any way unique.

Almost wherever distemper immunisation is practised, live egg-attenuated virus is used, and the procedure has by and large proved satisfactory. Nevertheless, a percentage of dogs fail to become immunised and eventually contract clinical distemper. Occurrences of this sort are causing concern, and, judging by most reports, they are commoner than hitherto. What are the reasons for the breakdown? A number can be listed; broadly they fall under 2 headings:—

(a) inadequate procedures;

(b) failure of the dog to respond.

Under (a) I would include inefficient techniques practised by the manufacturer or the veterinary surgeon. As to the former, every dose of vaccine issued should be capable of conferring protection; by this I mean that it should contain sufficient virus of an immunogenic strain properly preserved. The British Veterinary Codex states that each dose of vaccine should contain a minimum of 1,000 egg-infecting doses and that a dilution of 10^{-3} of the reconstituted vaccine must immunise the ferret to an experimental challenge. These standards are perfectly satisfactory, and all reputable firms will see that vaccine is tested batchwise in accordance with these recommendations. Living vaccines, in particular, are relatively unstable, and their deterioration may occur even under optimal conditions of storage. It therefore behoves the veterinary surgeon to see that his facilities for storage are adequate and that he so inoculates the vaccine as to leave no doubt. Here I must make the plea that the only sterile syringe is one that has been boiled. Chemical agents may be detrimental to distemper vaccine and, further, if inadequate, may result in infective agents being transmitted on contaminated syringes, particularly in busy surgeries; in this connexion Baker and Gillespie (1959) report that hepatitis virus is resistant to 95 per cent. alcohol for 24 hours.

The second reason for failure is the more important. We must realise that in vaccinating an animal we are introducing a biological active material into a biologically active host. Since no 2 animals are identical, we must expect some variation in response. With live vaccines, particularly, there is a distinct risk of interference from existing antibody passively acquired from the mother if vaccination is practised too early. After all, this is the basis of distemper-antibody assay, namely the ability of the animal's serum to neutralise the infectivity of the virus under laboratory conditions. Unfortunately circumstances preclude the preliminary evaluation of the antibody content of the puppy's serum; to overcome this, Baker and Gillespie (1958) have constructed a nomograph that predicts the optimal age at which to vaccinate, based on the titre of the mother. In a survey of breeding kennels, the highest titre of distemper-neutralising antibody we have recorded in a survey among bitches was 1:320, and it would appear from this nomograph that 7 to 8 weeks is the optimal age for vaccinating puppies. If, however, the kennels have had a recent history of distemper, it might be as well to withhold vaccination for 2 or 3 weeks longer. The other factor that may militate against successful immunisation is intercurrent infection with either virulent distemper or some other virus infection. We all know that vaccination in the face of infection is likely to end in disaster; but how many of us pause to think whether infection with other viruses can militate against a successful vaccination. For a living virus vaccine to be effective, the virus must multiply in the host, albeit to a restricted degree, but if a virus is to multiply it must invade the host cells; in this respect it is entirely different from a bacterium. If a virus does not enter the host cell, it will not multiply and it is well known to virologists that a virus already parasitising host cells may block the introduction of another virus. It is suggested that some of the failures to immunise dogs against distemper may be due to this—perhaps to blocking by hepatitis virus. In the survey mentioned above we were also struck by the relatively large numbers of bitches (20 per cent. or so) that had no antibody levels against canine distemper, or negligible levels of it, but relatively high titres to canine virus hepatitis, indicating recent or active infection with the latter. Most of these animals had a history of distemper vaccination, and it occurred to us to ask whether the presence of hepatitis virus under field conditions was in any way impairing distemper immunisation. There could be other explanations for this state of affairs, but recently an opportunity occurred to compare primary vaccination against canine distemper in kennels that

TABLE I
NEUTRALISING ANTIBODY RESPONSES TO PRIMARY DISTEMPER VACCINATION

Dog	Titre of neutralisation antibody to:		
	Hepatitis	Distemper	
		Pre-vaccination	28 days post vaccination
	Kennel "A"		
1	0	0	1:320
2	0	0	1:80
3	0	0	1:160
4	0	0	1:80
5	0	0	1:80
6	0	0	1:160
7	0	0	1:80
8	0	0	1:80
	Kennel "B"		
9	1:1,280	0	1:160
10	1:1,280	0	1:30
11	1:1,280	0	0
12	1:1,280	0	1:10
13	1:1,280	0	1:40
14	1:1,280	0	1:640

had either no evidence of hepatitis infection or evidence of recent or active infection. The levels of neutralising antibody achieved as a result of vaccination are shown in Table I.

It is generally agreed that a titre of 1:20 or less to distemper neutralising antibody indicates susceptibility; if this is so, two dogs at kennel B failed to immunise. It is appreciated that considerably more observations are necessary to establish a relationship of the kind suggested, and this is a line of enquiry we propose to pursue.

For a long time now we have been led to believe that the duration of immunity to distemper is lifelong. I question whether this is so in the absence of exposure to field virus, particularly as distemper in this country is now sporadic rather than enzootic and the chances of in-contact exposure to field virus are diminishing. At the recent conference in Madrid, and later, my colleagues and I have taken the opportunity of consulting a number of American practitioners on this point; for them it would appear that after about 3 years of age there is a distinct risk of distemper if a dog that has hitherto led a secluded life suddenly experiences infection, as for example on going to shows or kennels. I cannot but think that valuable animals exposed to risk at shows or kennels should be re-vaccinated 2 weeks or so beforehand. I encountered reports of the same sort of thing in Queensland last year, where dogs on farms normally lead a somewhat sheltered life, but are suddenly exposed to infection when cattle drovers with their attendant dogs pass through the neighhourhood. We can reasonably ask if country dogs over here are similarly at risk. Undoubtedly the level of antibody falls after vaccination; if it falls below 1/20 or so we believe that the animal is at risk. I know of no published work that shows clearly how long it takes to fall; to carry out such work would necessitate keeping dogs in strict isolation for a prolonged period. In our laboratories we are currently trying to elucidate this point. From our observations to date it appears that 6 months is too early for a booster vaccination, since by this time the antibody level to the primary vaccination has not fallen appreciably; possibly, therefore, 12 to 18 months will prove to be the best interval.

Hepatitis

I fully appreciate that the case for vaccination against hepatitis is controversial and I look forward to hearing the views of others on the subject, particularly of practising members. I think there is little doubt about the widespread incidence of the virus.

In a survey carried out by us up to August, 1959, that is, up to the time when the first living hepatitis vaccines appeared, we found that 46 per cent. of the blood samples we examined showed antibody titres in excess of 1:1280, which in our opinion indicates recent or active infection. A total of 89 samples were examined from 12 practices in various parts of the country; we were also interested to see whether there was any correlation between high titres in the bitch and a history of pre-weaning mortality in her puppies. There was a suggestion that this might be so. In this connexion we have had more recently an opportunity to examine the history of 36 bitches with a history of pre-weaning mortality extending from 1 to 4 pregnancies. Of these 26 had abnormally high titres of hepatitis-neutralising antibody, but 10 had low titres. I am not suggesting that the fading puppy syndrome is due to hepatitis infection, but it is a possibility we must not exclude, as suggested some years ago by workers at the Canine Research Station.

By and large hepatitis vaccines fall into 2 categories—the living and the dead. I will deal with the living first. Two independent groups of workers in the United States, those at Cornell and those at Pearl River, have developed attenuated strains of virus by passage through tissue-culture systems. Both pig and dog preparations were used. It appears that propagation in dog tissue was not sufficient to attenuate the strain, but that 10 or so subsequent passages in pig-kidney cells did produce attenuation, judged by the absence of clinical signs and *post-mortem* findings. The only untoward sequela that has been so far confirmed is a transient opacity of one or both of the eyes in a small percentage of vaccinated dogs. The strain developed at Cornell is now used in this country as a living hepatitis vaccine, and recently Piercy and Sellers (1960) have published work on its use in experimental dogs. It is clear, however, as pointed out by the authors, that the vaccine virus is excreted and indeed multiplies in contact animals. Further, from their paper it would seem that the antibody response in the in-contact dogs can be as good as that achieved by vaccination. The in-contact animals did not show any untoward sequelae, but I think that further work is needed before we can be certain on this point. Moreover, the fact that the virus is excreted in the urine suggests renal multiplication, and it will be some time before we can be sure that chronic renal damage has not been sustained. Experiments recently reported from the United States, have, however, been designed to show the safety of this attenuated strain (Cabasso, 1960). In these experiments the author carried out intraocular inoculations with both virulent and attenuated viruses. Of the 4 puppies inoculated with the attenuated virus, all developed a complete unilateral ocular opacity, which apparently persisted for some time. They did not, however, develop a febrile reaction or leucopenia, whereas of two comparable puppies given virulent material, one died though the other recovered. A similar experiment was carried out with histological findings at *post mortem*. No gross changes were observed in puppies given the attenuated strain, although mild degenerative changes were recorded in both kidney and liver. Four back passages in susceptible puppies failed to restore virulence.

We have also a ferret-cell adapted strain in use as a vaccine in this country, as well as non-attenuated virus instilled into the eyes as an oculentum. It is interesting to note that with the ferret-cell adapted strain it is claimed that vaccinated dogs do not become carriers or shedders of the virus.

As Fellows will appreciate, I am closely concerned with this problem, but rightly or wrongly believe that immunisation against hepatitis is of secondary importance to immunisation against distemper, and for this reason my colleagues and I have been greatly concerned as to whether interference with distemper immunisation can occur from hepatitis virus of one sort or another. We have reason to suspect that this may be so. In consequence, asking ourselves the question whether the alleged increased incidence of breakdown in distemper immunisation can be due in whole or in part to something of this kind.

TABLE II
TITRES OF NEUTRALISING ANTIBODY 28 DAYS AFTER VACCINATION WITH A COMBINED LIVING DISTEMPER/ HEPATITIS VACCINE

Dog	Titre neutralising antibody			
	Distemper		Hepatitis	
	Pre-vac.	Post vac.	Pre-vac.	Post vac.
1	0	1 : 80	0	1 : 40
2	0	1 : 5	0	1 : 1,280
3	0	1 : 160	0	1 : 80
4	0	1 : 40	0	1 : 40

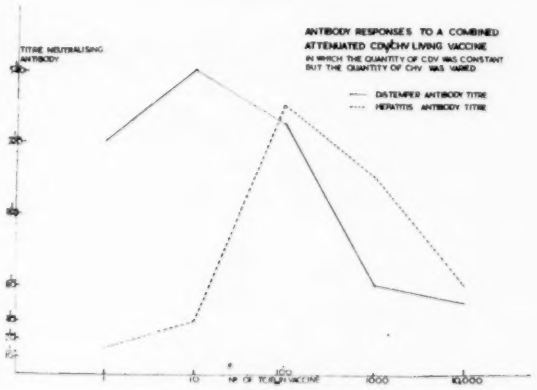
Table II shows the antibody levels achieved 28 days after vaccination in 4 comparable puppies vaccinated with a combined living distemper/hepatitis vaccine. It will be noted that one animal produced a relatively high hepatitis response, but a poor distemper response. These puppies were killed at the 28th day and *post-mortem* examinations were carried out. It seemed worthy of note that puppy No. 2 showed evidence of a focal interstitial nephritis similar to that described by Poppensiek and Baker (1951), though no inclusion bodies were observed (see Fig. 1).

In our laboratories we have carried out studies on a living distemper/hepatitis vaccine designed to show the optimal amount of each virus to include. The result of one such experiment is shown in Fig. 2. The experimental vaccines used all contained 5,000 Egg $I.D._{50}$ of distemper virus (Onderstepoort strain) and amounts varying from 1 to 10,000 tissue culture $I.D._{50}$ of hepatitis virus (Cornell strain). Each vaccine was inoculated into a pair of dogs. It will be seen that the optimal response was achieved with 1,000 $E.I.D._{50}$ of distemper virus and 100 $T.C.I.D._{50}$ of hepatitis virus. Further studies in this connexion are in progress, but clearly the possibility does exist of interference with distemper immunisation if too much hepatitis virus is included in the vaccine; further, too much hepatitis virus can apparently result in a less effective response. Our information is still far from adequate, and we are pursuing the matter actively at the present moment.

It is for reasons of the kind indicated that my colleagues and I have been reluctant to sponsor at this stage a living hepatitis vaccine. We have adopted the more cautious approach of attempting to immunise the puppy with a dead vaccine, to prevent acute hepatitis occurring in the young animal until it has had time to reinforce its natural immunity by exposure to field virus. The antibody responses to the dead vaccine, which must naturally be given in 2 doses, are adequate and satisfactory; since the vaccine is dead, it cannot be excreted, cannot possibly interfere with distemper immunisation, and it certainly does not provoke any side effects. In the absence of contact exposure, immunity naturally wanes, as indeed it does after all vaccines. We hold, however, that, since we are dealing with a virus by its nature likely to be ever present, and until we know more about the uses and effects of the living vaccine, there is some merit in a cautious and conservative approach. It could well be that our fears may prove groundless, but I think it behoves us as a profession to be alive to them and to be ever watchful.

A question often asked is what may be the right age of immunisation. It is believed that one can generally immunise against hepatitis earlier than against distemper, owing to the earlier disappearance of maternal antibody. One cannot help asking oneself whether this phenomenon is tied up with the ubiquity of hepatitis virus, as suggested by Montgomerie (1960). We can see no objection to giving a first dose of hepatitis vaccine at, say, 5 or 6 weeks of age and then simultaneous vaccination against distemper and hepatitis at the customary 7 to 8 weeks of age.

FIG. 2

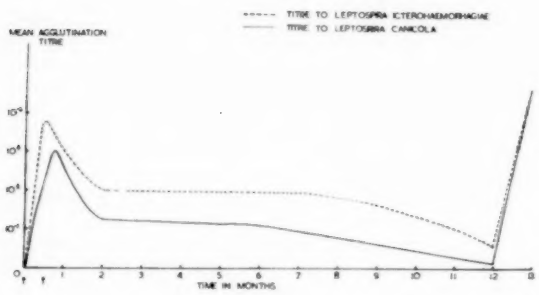

ANTIBODY RESPONSES TO A COMBINED ATTENUATED CDV/CHV LIVING VACCINE IN WHICH THE QUANTITY OF CDV WAS CONSTANT BUT THE QUANTITY OF CHV WAS VARIED
TITRE NEUTRALISING ANTIBODY
DISTEMPER ANTIBODY TITRE
HEPATITIS ANTIBODY TITRE
10
1000
10000

Leptospira

The effectiveness of Leptospira vaccines has now been accepted for a great many years, largely owing to the pioneer work of such investigators as Dalling, Okell and Broom. From work on laboratory animals it appears that the amount of agglutinating antibody is an indication of the degree of protection, and Fig. 3 shows the persistence of agglutinating antibody in dogs, together with the result of booster vaccination at 12 months. Last year the concept of combining both icterohaemorrhagiae and canicola vaccines was introduced into this country; similar vaccines have been in use in the United States for some time. The 2 antigens are compatible, and antibodies to both are provoked simultaneously; moreover, the vaccine can be given at the same time as distemper and hepatitis vaccines (Ablett, Uvarov, Addison & Baker, 1959).

FIG. 3

AGGLUTINATION TITRES FOLLOWING TWO INJECTIONS OF A COMBINED L.ICTEROHAEMORHAGIAE/L.CANICOLA VACCINE AND THE EFFECT OF A BOOSTER INJECTION 12 MONTHS LATER



Conclusion

In this general survey of the broad field, I have tried to focus attention on the points that seem to me the most controversial and to require elucidation. The introduction of new procedures, new methods of therapy, new prophylactics, invariably results in new problems arising. If we proceed blindly, we run a grave risk that canine immunisation may fall into disrepute: it behoves us, therefore, to be alive to the possibilities inherent in this situation and especially to look out for the unexpected.

Acknowledgments.—I am grateful to Dr. J. A. Baker and Dr. R. V. Johnson for permission to use their graphical results in presenting this paper, and to my colleagues, S. A. Keeble, R. E. Ablett, O. Uvarov, K. S. Chodnik and L. Baker for considerable assistance in its preparation, as also to Dr. D. A. Haig for supplying the strain of distemper virus and Dr. J. A. Baker for the attenuated strain of hepatitis virus used in our studies.

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The Opener's Remarks

Mr. S. F. J. Hodgman in opening the discussion said: Dr. Howell has given us a paper that is knowledgeable, informative, and courageous. I must tell you that I am almost entirely in agreement with everything he has said, and so, criticism being virtually impossible, I can only emphasise some of his points and ask him to elaborate others.

Some 12 to 18 months ago, in this very room, I heard an echo from my youth. People were saying, just as they said, 30 years ago, 'At last we have got the upper hand of distemper.' There is something ironic in the fact that we are here to-night discussing distemper all over again, but I am delighted by our seeming inconsistency, since I still hold that distemper is the canine disease of primary importance in this country. In some respects its clinical picture has changed over the last 20 or 30 years, and the neurological form seems to be more common to-day; yet instances of the old distemper picture are cropping up again, and we often hear the term "distemper-complex"—and "complex" is certainly the right word.

It has always impressed me as being strange that the dog should suffer from only 3 virus diseases, and in this connexion I should like to remind you that, for many years, canine virus hepatitis may have gone unrecognised as a disease distinct from distemper. May it not be possible that other virus diseases are still concealed under this heading?

I hope I shall not be suspected of inconsistency if, after the above remarks, I heartily endorse Dr. Howell's appeal that once and for all we abolish the misleading and unsatisfactory term "hard pad disease." This conveys to the lay mind a separate entity from distemper, and in fact until quite recently some veterinary surgeons were not too sure about it themselves.

A lot of water has flowed under the bridge since the days of the Laidlaw and Dunkin form of vaccination, but with it one did expect a 95 per cent. immunity which I, for one, believed to be life-long. The old method was far closer to a natural immunity than any practised to-day, and I hold that a naturally acquired immunity is usually life-long. The disadvantage of the old method was, of course, the 5 per cent. of failures. To-day we use the egg-adapted virus form of vaccination which is certainly safer; but it has its drawbacks, chief of which is that I do not believe it gives the lasting immunity conferred by the older method. I agree with Dr. Howell that this is particularly the case where dogs lead a normally secluded life; in such cases they may lose their immunity so rapidly that sudden exposure to infection may result in a breakdown after only 12 months. I believe that animals so isolated should be re-vaccinated after about 9 to 12 months.

On the other hand, in those cases where the dog comes up against natural infection during the period of immunity conferred by the egg-adapted virus, it would appear that the immunity lasts longer, and may possibly be for life.

In addition to any loss of immunity there are other causes for breakdowns. I do not propose to touch on the inadequate procedures referred to by the author as applied to the manufacture of these products; I want to stress these procedures in so far as they affect the practitioner.

I have always considered that any immunisation must be largely dependent on how material is handled and stored after it has been issued by the manufacturers; and the way it is kept by the practitioner before being used is obviously of great importance. The next point is proper sterilisation of syringes and needles. Admittedly the hepatitis virus is a pretty resistant one, but the distemper virus is not, therefore syringes and needles that have been sterilised chemically could affect the living vaccine.

One other point that I consider of even greater importance is the health of the animal to be vaccinated. I am absolutely convinced that it is completely wrong to inoculate any dog for active immunity if it has recently been in contact with infection or if it is not fit, and this prohibition applies to both distemper and C.V.H. If there is any doubt I consider vaccination should be delayed; but for reasons that are difficult to understand, sufficient importance is not paid to this necessary precaution, especially since the advent of the egg-adapted method of immunisation.

Another hazard against immunity response is, as Dr. Howell has said, the vaccination of young animals while they still have circulating maternal antibody. This risk is also incurred where the dog may have been given a passive immunity. While on the subject of immunity response I must refer to the very interesting paper recently produced by Piercy and Sellers. Dr. Howell has also referred to it but I should be most interested to hear him enlarge on this problem. After all, we often hear about "interference phenomena," and some of us have an idea of what it is supposed to be all about, but it is, I believe, still a subject of controversy amongst virologists, and certainly the aspect suggested by Dr. Howell is worth discussing more fully.

The essayist has said that vaccination against hepatitis is controversial, and with this I agree. I am a little doubtful as to why we are vaccinating against this disease, at least as extensively as we are doing. C.V.H., although widely

spread, has a comparatively low mortality rate though admittedly the morbidity is nigh. I suppose one can say that immunisation is justified if it will prevent damage to tissues; but as we all know dogs can have this disease without showing any clinical symptoms, and therefore unless the vaccination is done very early in life, it is difficult to assess its value. On the other hand if it is carried out early (and this is obviously the best time for it) can it interfere with the distemper vaccine? Should both be administered at, or about, the same time? In other words, can the "interference phenomena" operate in such way that one virus gives a better immunity response than the other, or even interferes with the immunity that would be conferred if one vaccination alone were administered? This inference may possibly be drawn from Piercy and Sellers' paper.

I am very interested in Dr. Howell's remarks concerning the carrier state of C.V.H. I gather that he considers an antibody titre in excess of 1:1280 indicates a carrier state. Would he please clarify this point as one would think that the higher the titre the higher the immunity.

The statement that he found a number of bitches with a high titre that had a history of puppy losses naturally interests me a great deal. Larin, you may remember, found similar results in a survey covering most counties in England, and he also found some connexion between C.V.H. and puppy mortality. I agree that much work is required on this subject.

One word on Leptospirosis. Will Dr. Howell tell us the position regarding the exportation of dogs which have been vaccinated (in so far as titres are concerned).

I cannot close without making some reference to the author's opening remarks on the modern way clients have of telling their veterinary advisers what they want and not asking for advice. Obviously, this is due to the extensive way in which commercial advertising is done to-day. This deplorable practice (by our clients) can be, and often is, very embarrassing and I shall be interested to hear the views of others.

The General Discussion

Dr. Howell: Two countries apply restrictions to the importation of dogs suspected of harbouring leptospirosis, namely Australia and Sweden. Australia will accept a dog with an agglutination titre of less than 1:100, and Sweden will accept evidence that there is no rise of titre indicative of active infection.

As regards hepatitis antibody levels in relation to immunity and the carrier state, much work is required to clarify this point. It involves not only serum examination but frequent urine sampling for the presence or absence of virus by tissue culture titration; all of which is costly and time consuming work. Our present assumptions are based on field evidence, history of the kennels in question and analogy. As far as distemper is concerned I think we are all agreed that the virus is not harboured by the dog, but with hepatitis the virus is harboured for 1 or 2 years, and perhaps even longer as judged by work reported from the United States. With hepatitis we found 46 per cent. of dogs in our survey had very high titres in excess of 1:1280, which we consider indicates recent or active infection.

The interference phenomenon is well known in virology. It was a legitimate question for us to ask ourselves when contemplating vaccination with 2 living viruses, particularly with 2 that have, by and large, the same tissue affinity in the dog. As I said in my paper, our fears on this point may be proved groundless, but it is a point on which we must be satisfied.

Mr. Taylor (South Kensington): May I ask the speaker, whether he approves of administering serum to a very young animal exposed to infection, in order to produce an immediate immunity, and if so, how long after this would he consider it wise to give the vaccine?

Dr. Howell: Passive immunisation with serum is an acceptable and rational method of immunising young animals against diseases to which they are likely to be exposed shortly after birth. The question, however, as to whether the young animal's antibody-forming mechanism is functional at birth is a debatable point, and as far as I am aware no work has been carried out to elucidate this point in the case of the dog; neither is there any evidence as to how long passive immunity lasts after giving serum but I would suggest two or three weeks at the most. I do not favour giving serum and vaccine at the same time.

Mr. Poole (Weybridge): I wonder if we could be quite clear when we are generalising and when specialising in what has been said regarding distemper virus we are referring to reactions to a live virus vaccine. Am I wrong in inferring that reactions to other types of live virus distemper vaccines are not necessarily the same?

Dr. Howell: To the best of my knowledge the only distemper vaccines now in genereal use, with few exceptions, are based on the egg-adapted virus and my remarks apply to that form of vaccine. I am afraid I have no experience of distemper vaccines other than the egg-adapted. By and large all those developed produce the same sort of response. The only hazard is if they are over-adapted, as recent work from Onderstepoort and the United States suggest.

Mr. Manscombe (West Wickham, Cambridge): I would like to put a hypothetical question to Dr. Howell: if I am presented with kennels in which there is a high titre to hepatitis in the bitches and a pre-weaning mortality, am I justified in assuming that surviving puppies will be immune or should I undertake hepatitis vaccination?

Mr. Forbes (Romford): I had a case yesterday of a client who wanted to sell 3 puppies from a litter, of which several had died within 24 hours of birth, and who had previously lost 2 complete litters. She asked us whether she was safe to sell the surviving pups. Is it possible that these pups have developed an immunity but are also actively excreting the virus? If so is there any means whereby we can test these puppies to see whether they are safe for sale?

Dr. Howell: This is a good question. If the bitch has a high level of antibody the puppy will undoubtedly be born with a high level also. There will, however, be a short period during which its passively acquired immunity is of a low order and before it has a chance to develop active immunity as a result of exposure to natural virus. It would seem that passively acquired antibodies to hepatitis can be eliminated fairly rapidly—3 weeks or so—possibly as a result of exposure, from the moment of birth, to virus being harboured and excreted by its mother. Whether one should vaccinate puppies under these circumstances is a debatable point. I, myself, feel that one has to play for safety, and at least no harm will be done by vaccinating such animals.

Mr. Bowden (Animal Health Trust, Kennett): If, as I believe you stated, the hepatitis virus is ubiquitous, can it exist in the carrier state without there being some tissue damage? And to come to virus vaccine, if the virus must multiply as a living virus in order to confer immunity, can it multiply without causing damage?

Dr. Howell: If a virus is excreted in the urine it strongly suggests that renal multiplication is taking place. This could take place in the presence of circulating antibody due to the somewhat peculiar blood supply of the kidney. Excreted virus can be picked up again by the dog and continue to boost its antibody level. A virus need not necessarily produce pathological tissue damage as evidence of multiplication, indeed, there would appear to be, at least in pigs and man, a whole host of symbiotic viruses situated in the alimentary tract and which apparently produce no disturbances at all. The strain used in a living virus vaccine must multiply in the animal but apparently does not produce a change that we can recognise.

Mr. John Bywater (Sandwich): You mentioned that with live hepatitis vaccine it is possible for a vaccinated animal to excrete virus and for the virus to be picked up by an in-contact animal. Is there any evidence that by further passage of that material through other animals there can be any reversion to original virulence? And, referring to egg-adapted distemper viruses, is there any evidence that the virus is excreted, and that after further passages in-contact animals may revert to virulence?

Dr. Howell: All authors to date freely admit that the pig cell attenuated strain of hepatitis virus is excreted. So far, the only published work on back passage in dogs has involved 4 passages, and no evidence of reversion occurred. One would like to see many more passages done

—say 10 to 15. As far as the egg-adapted strain of distemper is concerned there is no evidence of excretion.

Dr. Appleby: I should like to ask Dr. Howell a question arising out of a remark that the distemper virus does not persist for a very long time in a dog in which, however, we find inclusion bodies. Can you tell us anything about the period of infection when these first appear, their frequency in relation to the disease in general, and their persistence within the tissues within the living animal?

Dr. Howell: I am sorry to admit defeat on this question; perhaps someone else could help?

Dr. S. E. Piercy (Beckenham): Dr. Howell started off on the premise that the reported numbers of breakdowns, through vaccination and failures to vaccinate efficiently, were on the increase. This may be so, but I was rather hoping that we would hear a comment on this point from the practitioners present. Like other commercial firms, we at Beckenham keep a very close watch on this question, and any reports or breakdowns or trouble with our own product are carefully examined and recorded. A recent examination of these records has revealed that the number of adverse reports received during the past 12 months has not, in fact, increased and remains very small. I wonder, therefore, just how accurate this suggestion is, that there are more breakdowns and failures to vaccinate efficiently to-day?

The whole question of immunity in the dog from vaccination is a little confused because laboratory workers usually interpret their results on the level of demonstrable neutralising antibody present. Techniques vary from one laboratory to another, and unless precisely the same technique is used by all laboratories you simply cannot compare results with accuracy. For example, I should like to ask Dr. Howell if he would tell us, in the experiments he described, how many tissue culture infecting doses of distemper virus were used in his neutralisation tests and then perhaps we can compare his results with others.

The Cornell workers—and they have really led the field in research into distemper and hepatitis during the past 5 years—have worked out, according to their techniques, that if the serum antibody level drops below 1 in 30, a second dose of vaccine will boost the antibodies, and they therefore say that that dog is susceptible. Now I think that is a wrong statement. The dog is obviously capable of response to a second vaccination and the antibodies will rise, but it does not necessarily mean that that dog will contract natural infection and show clinical disease and either recover or die; that is a very different thing. I think that is an important point to remember when we are talking about how long immunity lasts.

It is perfectly true that in a percentage of dogs the neutralising antibody level falls after 12 months. In some it will have fallen to this level of 20 or 30. In others it will take two years for that level to be reached, and in a further percentage it will take longer. There is no doubt there is considerable variation in the individual response of particular dogs, but does it means, as I say, that if a dog's antibody level has dropped to 1 in 30 it will contract clinical distemper if it is exposed to a natural challenge, or does it simply mean it is capable of having its antibodies boosted? I think there is quite a difference there.

Duration of immunity—I entirely agree with Dr. Howell: we do not know how long dogs will remain solidly immune after vaccination in the absence of renewed contact with the disease, and all claims for permanent immunity of any vaccine are made on the assumption that in nature, in animals leading normal lives, they will come into contact with the disease sooner or later and have their immunity reinforced.

I do agree that it may well be that a proportion of dogs in this country, now that the incidence of distemper has dropped so much, may be becoming susceptible again, and it could be that revaccination in these cases is necessary. The trouble is that the only way to find out with any certainty is to bleed these dogs and examine their sera for the presence of antibodies. I would very much like to see a scheme set on foot whereby some kind of survey, possibly in selected areas, could be done whereby the sera of dogs thought not to have been in contact with the disease since vaccination could be analysed. I think it would be a most interesting and valuable thing to do.

A point that really interests me is Dr. Howell's suggestion that there may be cross-interference between the 2 viruses of hepatitis and distemper. He has produced certain figures, which must be accepted, naturally; they are, as he says himself, from only 4 dogs here and 4 dogs there. There are plenty of figures, of course, of other people's experiments—including our own—which show just the reverse, namely that if you give dogs the combined vaccine containing both living distemper and living hepatitis viruses they will respond and produce antibodies against both viruses. In fact, the whole concept of a combined living vaccine stemmed from the finding that dogs could contract both distemper and hepatitis simultaneously, and as soon as that was noticed the idea that possibly a combined vaccine might be elaborated was pursued. What I do not quite understand is this: I accept what Dr. Howell says, that a virus, in order to stimulate antibody response immunity, must multiply in the animal and occupy its cells; that is a well accepted view. Now if, either by simultaneous natural infection or by inoculating the 2 viruses together as a vaccine, it is possible for them to live together happily and multiply, as undoubtedly they do, why should it follow that it is any more difficult than it was for distemper virus given in the vaccine to enter the system and occupy the cells it prefers in dogs that are already immune to hepatitis?

Dr. Howell: Perhaps I may deal with your specific question first. We employ 300 to 500 $E.I.D._{50}$ in 0.1 c.c. in the case of distemper; as far as hepatitis is concerned we use 500 $T.C.I.D._{50}$ in 0.2 c.c.—which is about 5 times more than employed in your laboratory. I find it very pleasing and gratifying that another group of workers have come to the same conclusion with regard to distemper immunisation. the whole thing depends on contact rate with field virus. The question of what levels of neutralising antibody indicates susceptibility is difficult to say, particularly under field conditions, but in the laboratory it appears to be 1:20 to 1:30.

On the question of breakdowns, my remarks were based on opinions expressed by practising veterinary surgeons in various parts of the world and it would be interesting to hear something from practising colleagues on this point.

As regards interference, I endeavoured in my paper to be very careful, saying that this was a possibility that must be examined. My feeling is that in the published work to date insufficient dogs have been included. Our observations, as reported, were part of a wider experiment to study the effectiveness and safety of living hepatitis vaccines, and the 4 reported were part of this. These 4 had been injected with a combined distemper/hepatitis vaccine and we were indeed surprised with the observations as reported. My point in mentioning it was that this is the sort of thing we must look for, since, even if it occurs in only one or two instances, it is important to the veterinary surgeon and to the dog owner.

Mr. Fairweather (Ilford): There are, I remember, only 2 cases in 5 years that have come back as definite breakdowns. Both were reported and we did feel slight anxiety at the time. I practice in the East End of London—where they are quite keen on inoculation—and we inoculate up to 100 dogs a month. I am quite sure that they would certainly come back and tell us if they had breakdowns.

Dr. Howell: In my remarks on distemper immunisation you will recall I said that, by and large, the use of egg-adapted virus is very successful, and it has been adopted as the standard method of vaccination in most countries of the world. Quite obviously, if it was not satisfactory it would have been dropped long ago. However, it is the small percentage of dogs which apparently fail to immunise which provoke much interest and which must be a matter of concern to us all.

Mr. Hodgman: Recently I had cause to go to the Battersea Dogs' Home and I saw an enormous amount of so-called old-fashioned distemper—eye discharge, emaciation, etc.—and I think that anybody could recognise it as absolutely typical. They also had quite a number of dogs which were semiparalysed, which they themselves—laymen, I must admit—diagnosed as suffering from hard pad disease. I have the impression that one gets more of these breakdowns in country districts among gun-dogs, foxhounds,

and in general the type of animal which does not normally come up against infection and get a "booster" like the town dog. I think vaccination has done remarkably well; but it does not alter the fact that we are getting breakdowns—in the country districts quite commonly.

Dr. Howell: I would like to add to Mr. Hodgman's remarks. The widespread and effective use of distemper vaccination has resulted in a markedly reduced incidence of field virus in the last few years. In consequence, the possibility of an incontact boost is less likely, and our feeling is the same as Mr. Hodgman's: namely that more distemper is cropping up in older dogs leading a somewhat restricted life.

Mr. Turner (Northolt): I would like to ask Dr. Howell exactly how he defines a true breakdown compared with an incubation breakdown. What period of time should elapse after inoculation before he would be convinced that a dog showed signs of distemper due to a breakdown and not due to the fact that it was incubating virus at the time of the inoculation?

Dr. Howell: Fourteen days is a reasonable figure, and if distemper occurred with that time I would suspect that the dog was, in fact, incubating the disease pre-vaccination.

Dr. Piercy: I have not quite understood what Dr. Howell means. If, for example, a dog is vaccinated the day after it has come into contact with natural infection—in other words, it is incubating the disease by 24 hours—it might well be 3 to 4 weeks before the owner notices obvious signs of distemper. Distemper varies, as we all know. The primary temperature rise would almost certainly be missed and the secondary temperature rise would not occur for another week or more. Secondary symptoms might be slight and might not be put down to distemper. Only, shall we say, 5 or 6 weeks later when neurological symptoms developed would the owner really realise something had gone wrong. I am sure you did not mean a fortnight in that sense?

Dr. Howell: Dr. Piercey is probably quite right if the form of distemper is primarily neurological, and I thank him for his intervention.

Professor McCunn: It has always been in my mind that when you vaccinate you have to have co-operation between the vaccine and the animal receiving it. There are many animals that just will not "take"; that is equally well known in vaccination for smallpox, etc. We all know that antitetanic serum is pretty well 100 per cent. certain if you give the appropriate dose soon after the wound is received; yet I have had cases in which it failed. You are bound to have occasional breakdowns, because of some fault in the inoculum or because of some fault on the part of the receiver. You have no means of testing the particular dose of serum used. You can only test the batch from which it came if there is any left. We all know that the incidence of distemper has been greatly reduced. It began to be reduced at the Laidlaw Dunkin time, when the common form of distemper—what Mr. Hodgman called old-fashioned distemper—was of the pulmonary and the alimentary type. The neurological form of distemper was just an odd thing one saw occasionally. I think that the egg-adapted virus has been of great assistance; I see a number of dogs and I talk to many people: I do not think there are many breakdowns. I think that the breakdowns that do occur are probably something inherently at fault in the dog itself, or it may be that the inoculum dose was not up to standard. I do not think that the problem needs any great scientific explanation.

Dr. Howell: I would always agree with Professor McCunn, and as I said earlier, if one introduces a biologically active material into a biologically active host one must expect a variation in response. We all know that some animals fail to respond, but as far as the dog is concerned what we have endeavoured to do this evening is to pin-point some of the reasons for occurrences of this sort.

Dr. R. F. Sellers (Beckenham): I was interested in Dr. Howell's preliminary finding of interference with the antibody response to distemper in the presence of high level hepatitis antibody and hepatitis virus; but does not hepatitis virus multiply in certain organs of the body and distemper in others, and could this interference be a toxic effect or something similar? Again we know that if animals are depleted of protein their immune response is very poor.

The important point, I feel, about distemper and hepatitis vaccination, is, as you mentioned before, the conversion from a passive immunity passed on from the bitch to an active immunity given by the vaccine, and in that respect I feel that the published literature tends to simplify the problem. We have examined the serum antibody levels in 4-week-old puppies from vaccinated bitches, and from bitches already having virus antibody levels, and have found quite a variation in the antibody levels in the puppies even among those from the same litter. This suggests that puppies must become susceptible to the diseases at different times, which again makes the specification of the time of vaccination difficult. The art of vaccinating, then, appears to be getting the vaccine in at the correct moment before the virulent virus can get in itself.

Another point Dr. Howell made was that he felt rather cautious in using an attenuated hepatitis virus. As you know, we have been using this at Beckenham and in our first experiment, in dogs that received the combined vaccine followed by challenge first with virulent distemper and then with virulent hepatitis virus, we found no significant histological changes in the kidneys. In the second dual vaccine experiment, which is to be reported later, where 24 days after vaccination we challenged with virulent distemper and hepatitis simultaneously, the histological picture is not yet completely analysed, but there does appear to be a slight change. However, we have seen no change in kidneys of dogs given the vaccine alone. The attenuated hepatitis virus was excreted from the kidney and spread to dogs in contact as was also found by the workers at Cornell and Pearl River, but we feel that is still safe. It is noteworthy that no one has yet been able to passage the excreted virus beyond 4 dog-to-dog passages, whereas with the virulent hepatitis one would suppose it possible to go on indefinitely.

From our own and other workers' surveys, it is generally agreed that virulent hepatitis virus is widespread in this country. We, therefore, feel that it is better to have an avirulent virus around the place rather than the virulent hepatitis virus. I would prefer my dog, if it went along the street, to pick up attenuated hepatitis virus from a lamp post rather than the other kind.

Finally, I would like to ask Dr. Howell if in his experiments with inactivated hepatitis vaccine and after challenge with hepatitis virus he has made a histological examination of the organs?

Dr. Howell: The answer to Dr. Seller's last question is "yes," and we could find no evidence of contraction of the disease in dogs challenged a fairly short time (28 days or so) after vaccination. We have not made later studies.

I would agree entirely with Dr. Sellers on the optimal time for vaccinating puppies. As regards failure to immunise being due to a toxic factor, I cannot comment upon this as many things are possible and clearly malnutrition could well be taken into account.

Miss Uvarov questioned the value of clinical diagnosis in distemper breakdowns. She asked Mr. Hodgman if he had come across a distemper-like syndrome caused by *Klebsiella*, which had recently been reported in America (Gianforte, E. M., & Brown, R. G. (1959). *Mod. Vet Pract.* **40**, 64). The condition was clinically similar to distemper but the only organism isolated from affected animals was a *Klebsiella* sp. Experimentally it was pathogenic to puppies when injected intracerebrally, in animals susceptible and immune to distemper she encountered only one similar case in an Alsatian puppy immunised previously to distemper. The clinical symptoms included all those found in the terminal stages of distemper including hardening of the pads, chorea, and fits. Extensive laboratory examination was carried out and only *Klebsiella* spp. was recovered. Distemper virus *was not* isolated.

Mr. Hodgman's question as to why we vaccinate against hepatitis had not been answered. There was now some evidence that nephritis could be present as a result of canine virus hepatitis. By vaccination against this disease, the veterinary surgeon was trying to prevent the acute

culminating type of virus hepatitis, which occurs in a proportion of cases, but may also be safeguarding some of his patients from nephritis due to this virus. Further work, however, was necessary to verify this aspect of virus hepatitis.

Miss Uvarov asked Dr. Sellers to elaborate on his statement that "with attenuated hepatitis vaccine there was no significant change in the kidneys," because she was not very happy with this meaning of *significant* or *not significant;* what did it mean in relation to the pathological state?

Mr. Hodgman : In the days when I was in practice I had very little difficulty in making a diagnosis of distemper; any intelligent lay man could recognise it. To-day, however, I entirely agree that it is an entirely different matter, particularly in the early stages.

A young animal, unvaccinated, may be brought into the surgery with a temperature and a history that it is off its food. Perhaps the only sign one can detect is a tonsillitis. With such a history it is obviously very difficult to differentiate between distemper and canine virus hepatitis. In the old days one got a rise in temperature on about the fourth or fifth day following infection either naturally or artificially produced, and this rise in temperature would occur with monotonous regularity. I inoculated my own dogs by administering a virus and then waiting for the initial rise in temperature, when I gave serum intravenously. I did quite a considerable number in this way, and never had a failure. Secondary invaders usually manifested themselves about 14 days after the initial rise in temperature.

To-day, should I get a case presented to me in the early stages I am seldom dogmatic, but warn the owner that the trouble may be either distemper or virus hepatitis. I advise that the dog should be confined and exercise cut out, as I think this is most important. I am absolutely sure that if dogs in the early stages of distemper are allowed to live a normal existence, then they are much more likely to develop severe neurological symptoms than if they are kept quiet. After all, this is what happens in cases of polio.

Dr. Howell : I agree: could we not hear from practising members?

Miss Welch : It has been said to-night that general practitioners are not giving enough help to research workers. I would like to suggest a field trial consisting of a test for antibodies in blood samples from a percentage of all the dogs we immunise against distemper, sampling to be done at 1 to 2 years or more.

We have been asked about breakdowns. Since using egg-adapted virus I have had only 2; both were under a year old, and both were serious cases. They died, or were destroyed, and a thorough pathological investigation was made.

I am getting typical chronic nephritis in dogs which are now old, which I treated many years ago for distemper. Some of them made apparently good recoveries and I have not seen them in the meantime; so far as I can gather they have not been ill since. I wonder whether they had hepatitis and/or distemper?

I would like to ask Dr. Howell these questions: if we are requested to immunise older dogs against hepatitis—as is happening with this fox scare—what risk do we run of triggering off a sub-clinical infection? If one is asked to immunise a young puppy which has come from kennels where distemper serum of dubious manufacture has been given, what interval should one allow before giving that puppy vaccine?

I am rather a doubting Thomas over this idea of boosting an artificially immunised animal by letting it come into contact with natural infection. I have seen so many dogs of 4 to 9 years old with typical distemper, or whatever you like to call it, and most of them have had adequate opportunity to go on boosting up their natural immunity. They have never been vaccinated, have lived in areas where the disease was rife, and they have reached an age where one would expect them to have an unassailable immunity. Most of these dogs have either died or been destroyed. I think that is a point against the theory of boosting up artificial immunity to distemper.

Dr. Howell : With regard to possible hazards in vaccinating older dogs against hepatitis in the case of the "fox affair" I am not suggesting that there is any connexion between the two; there was a certain amount of evidence when the condition first appeared, by analogy from what happened in the United States, but so far there has been no clear evidence to substantiate it. There is apparently little risk in vaccinating older dogs.

Mr. Keeble : I would like to make one point regarding something Dr. Piercy and Dr Sellers said: they have rather suggested that since hepatitis and distemper have slightly different tissue affinities it is unlikely that a carrier of hepatitis would be unable to respond to distemper vaccine. Our point is that in the carrier dog there is a large amount of virus being circulated in the body and there is a great chance that all the antibody-producing cells in that dog will be swamped with this virus. It is a well-known fact that antibody-producing cells are able to produce only one antibody at a time, and if these cells are swamped with one particular antigen, *i.e.* hepatitis, the dog is going to have a much reduced chance of producing a good antibody to distemper.

On this matter of inclusions, it is possible to demonstrate by immunological methods that they are specific for the particular virus concerned and that they usually occur at the height of the disease. It would be an extremely difficult process to examine a series of dogs over a long period of time to find out just how long these inclusions do survive, and I am quite sure that at the moment there is no report in the literature on long-term studies of this sort.

Thanks to the Speaker

The Chairman (Mr. G. L. B. Henderson) : Dr. Howell, I would like you to know how grateful we are for your appearance here to-night. It is obvious that you have spent much time preparing your paper, and in translating research language into practical terms which people like myself can understand. We all appreciate that very much. I would also like to thank Mr. Hodgman for opening the discussion in so stimulating a fashion.

AFRICAN HORSE SICKNESS AND SWINE FEVER

The rapid spread and disastrous effects of African horse sickness in the countries of the Near East, the Mediterranean Basin and the Indian Sub-Continent, as well as the appearance of African swine fever in countries of the Western Mediterranean and Southern Europe, have created an emergency situation requiring strong and rapid action from all governments concerned, states a message from FAO in Rome.

The Food and Agriculture Organisation, in consultation with the International Office of Epizootics, convened an emergency meeting in Paris from January 17th to 20th to plan veterinary sanitary measures on a national, regional and international scale for the establishment of an efficient control campaign.

Frosts had killed the insect carrier of horse sickness during the first winter months and had temporarily stopped the infection in the Near East, but it was almost certain that the disease would re-appear in the spring unless mass vaccination was carried out.

In the case of African swine fever, the virus has been identified and intensive research is under way in Portugal and Spain as well as in East African countries to develop a vaccine, but unfortunately little progress has been made so far. Slaughter policies, therefore, remain the major weapon for the control of this disease in the view of FAO officials.

Factors Predisposing to Canine External Otitis

BY

GORDON FRASER

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University of Edinburgh**

SUMMARY.—There was an increased breed susceptibility to otitis in Spaniels, Miniature Poodles and some other breeds with overlapping ears.

An important predisposing factor was the presence of a clinical skin disease in 38 per cent. of the affected dogs. Extension of the skin condition to the lining epithelium of the external acoustic meatus may frequently give rise to the initial lesion of otitis.

Ear mites were seldom numerous in affected ears and only 6 per cent. of dogs were clinically infested.

Bacteria and fungi were not considered to be important causal agents of ear disease in dogs.

Introduction

MANY factors are said to predispose the canine external ear to otitis and it is generally agreed that there is an increased susceptibility to otitis in the long-haired, pendulous-eared breeds such as the Cocker Spaniel and Miniature Poodle (Joshua, 1958).

Trauma, often associated with faulty cleaning of the ears; nervous disorders, endocrine imbalance, allergy or the presence of excessive moisture or tumours within the auditory canal, greatly favour the onset of otitis. Additional predisposing factors include generalised infections and diseases such as distemper, the complications of which may give rise to pustular lesions in the external ear canal (Kirk, 1948; Kral & Novak, 1953).

The aetiological importance of ear-mites (*Otodectes cyanotis*) has been stressed by a number of workers including McGinnis and England (1949) and Jennings (1953), whereas Joshua (1958) is of the opinion that the parasite is a rarity in the canine ear and that suspected cases of parasitic otitis seldom respond to acaricidal dressings.

Some workers consider that bacteria and fungi are chiefly responsible for the onset of the disease, the most important species being *Proteus, Pseudomonas,* staphylococci, haemolytic streptococci, yeasts and fungi, while others assert that bacterial infection is always secondary to some other factor. On the other hand Gustafson (1955) stressed the importance of asporogenous yeasts and claimed to have successfully induced the clinical condition in healthy dogs by instilling cultures of *Pityrosporum canis* into the intact external acoustic meatus.

During a detailed study of the bacterial and fungal flora of the external ears of dogs suffering from otitis (Fraser, 1957), it was noticed that many dogs were also clinically affected either with a skin complaint, ectoparasitic infestation or some other factor which may have predisposed the ear to otitis; and it is the purpose of this paper to discuss these findings.

Methods

Two groups of affected dogs were studied, the first (Group A) consisting of 351 animals which were examined before treatment and the second (Group B) of 100 dogs which failed to respond promptly to treatment and were examined before, during and after treatment. The case history of each dog was examined for evidence that it was also clinically affected with one of the following conditions within a period of 2 months prior to the onset of otitis.

Skin lesions: including dermatitis, pruritus, eczema, conjunctivitis, pedal eczema and anal adenitis.

Ectoparasitic infestation: animals clinically affected with lice, fleas or ear mites. A few cases of parasitic mange were also included where it was not possible to identify the causal agent with certainty.

Foreign bodies: excessive debris, such as small stones and grass awns, warts, various tumours and trauma.

Debilitating conditions: generalised infections including distemper and hard pad were classified with other commonly occurring conditions such as tonsillitis, nephritis and "fits."

No history: these dogs were apparently unaffected by any of the factors included in the four previous groups and, apart from otitis, were believed to be clinically healthy.

Results

Age, Sex and Breed Susceptibilities

As detailed information regarding the age, sex and breeds of the general dog population were not available during the earlier part of this survey, the possibility of a breed predisposition to otitis could only be ascertained by classifying the 351 unselected cases of otitis by their breeds and comparing the results with those of 100 dogs which failed to respond promptly to treatment (Table I).

The breed distribution figures in Table I show that Spaniels accounted for over one third of the dogs suffering from otitis, and outnumbered any other breed by approximately four to one. Collies, on the other hand, were the most popular breed during the period of the survey, but represented only 9.1 and 3.0 per cent., respectively, of the affected dogs in both groups.

Apart from Spaniels and possibly Border terriers, there was little evidence that other breeds were more prone to the more chronic forms of otitis.

As the above findings do not take into account local breed popularities, recent trends in fashion, and other factors which may influence the apparent breed susceptibilities to otitis, the survey was extended to include all dogs examined in the Small

TABLE I
THE BREED DISTRIBUTION OF DOGS WITH OTITIS

Group A. —351 unselected cases of otitis
Group B. —100 dogs which failed to respond to treatment

Breed	Group A	Group B	Breed	Group A	Group B
Spaniel ...	34·8%	42·0%	Dachshund	1·7%	3·0%
Labrador ...	8·3	10·0	Bulldog	1·1	—
Collie	9·1	3·0	Bull Terrier	1·1	2·0
Alsatian... ...	6·3	4·0	Airedale	1·1	1·0
Scottish Terrier	4·8	6·0	Sealyham	1·1	—
Cairn Terrier ...	3·4	—	Corgi	0·8	1·0
Boxer	3·1	2·0	Chow Chow	0·8	—
Poodle	3·1	3·0	Pekingese	0·3	1·0
Fox Terrier ...	2·8	1·0	Yorkshire Terrier	0·3	—
West Highland Terrier ...	2·5	3·0	Kerry Blue Terrier	0·3	—
Border Terrier ...	1·9	6·0	Greyhound	0·3	—

Mongrels and other unidentified breeds are not shown.

Animal Clinic of this School during the 6 months' period from October, 1959, in an attempt to obtain some estimate of the breed incidence to otitis (Table II). The results also include a "Sample Normal Population," the annual figures being obtained from all dogs examined at the School's clinic since January, 1957, whatever the reason for the examination.

TABLE II
THE BREED INCIDENCE OF DOGS TO OTITIS
BREED DISTRIBUTION

Breed	General population ("Sample Normal Population") 1957 (5,678)	1958 (6,075)	1959 (6,652)	1960 (3,120)	Otitic group 1959—60 Number of dogs affected/examined (293/3,120)	Breed incidence (9·4%)
Miniature Poodle	2·7%	5·5%	6·5%	7·1%	54/221	24·4%
Spaniel...	12·2	9·1	8·1	7·8	55/244	22·7
West Highland Terrier	2·1	2·6	1·2	2·6	14/82	17·1
Wire Haired Terrier	3·5	0·4	0·5	0·7	7/50	14·0
Scottish Terrier	3·9	1·5	1·7	2·0	8/61	13·1
Labrador	8·6	7·3	7·2	8·5	29/266	10·9
Pekingese	2·4	2·2	2·2	1·9	6/58	10·3
Alsatian	7·9	8·4	8·1	7·5	20/236	8·4
Corgi	2·0	1·8	2·4	2·4	6/74	8·1
Border Terrier	1·7	1·6	1·5	1·3	3/42	7·1
Collie	13·6	15·4	14·0	13·9	30/433	6·9
Dachshund	1·4	1·7	1·9	1·6	3/49	6·1
Cairn Terrier	4·5	5·0	4·9	4·8	6/151	4·0
Boxer	2·4	2·9	3·9	4·4	3/137	2·2
Greyhound	3·7	3·9	4·3	4·6	2/145	1·3

Although an apparent increased breed susceptibility to otitis may have been due, in some cases, to the small number of dogs examined, the results in Table II appeared to suggest that Spaniels, Miniature Poodles and certain Terrier breeds were especially prone to ear disease. Due to their greatly increased popularity in recent years, the number of Miniature Poodles in the otitic groups increased from 3.1 in 1957, to 18.1 per cent. in 1960, while the breed incidence figure of 22.4 per cent. was the highest of all the breeds examined. Greyhounds, on the other hand, were seldom affected and of the 145 examined only 2 were suffering from otitis.

More detailed findings showed that the mean age of all affected dogs was 5.3 years, compared with 4.3 years for healthy dogs; that the ratio of males to females was 1.65 to 1, whether or not otitis was present; and that no fewer than 68 per cent. of affected dogs had overlapping ears.

Factors Predisposing to Otitis

The various factors which may have predisposed the external ears to otitis were classified, for convenience, as "skin lesions," "ectoparasitic infestation" and "all other factors," and the findings were summarised as shown in Table III.

TABLE III
FACTORS PREDISPOSING TO OTITIS

	Number of dogs clinically affected		
	Skin lesions	Ectoparasites	Other factors
Group A (351 dogs)	134 (38·2%)	45 (12·5%)	50 (14·3%)
Group B (100 dogs)	59	14	10

The results in Table III show that the incidence of ectoparasitic infestation and other predisposing factors in both groups of dogs suffering from otitis was similar, and that at least 1 in every 3 of the unselected cases of otitis (Group A) were also clinically affected with a skin complaint. Moreover, in dogs which failed to respond promptly to treatment (Group B) the incidence of clinical skin disease was almost 60 per cent. More detailed findings showed that of 351 animals in Group A, 11 per cent. were suffering from conjunctivitis, 7 per cent. from anal adenitis, 8 per cent. from pedal eczema and 31 per cent. from other forms of skin disease. The figures for the 100 dogs in Group B were proportionately very similar.

Ectoparasitic infestation with ear mites, lice and fleas accounted for 6, 3, and 2 per cent. respectively

of the dogs in Group A, and 6, 2, and 5 per cent. respectively of the dogs in Group B. The commonest conditions included under the heading of "other factors" were distemper in 6 per cent. and nephritis in 4 per cent. of all dogs suffering from otitis.

The Bacterial Flora

Although the bacterial and fungal flora of the external acoustic meatus is probably of little aetiological importance in canine external otitis (Fraser, 1957; 1960), an attempt was made to correlate the predisposing factors with the more frequently occurring micro-organisms in infected external ears. The results of this investigation are summarised in Table IV, which also includes, as controls, the results of an examination of the external ears of 35 clinically healthy dogs.

TABLE IV

CORRELATION OF THE BACTERIAL FINDINGS WITH THE MORE IMPORTANT PREDISPOSING FACTORS TO OTITIS

Species	Healthy animals (35 dogs)	Group A (351 unselected cases of otitis)			Group B (100 chronically affected dogs)		
		History free	Ectoparasites	Skin lesions	History free	Ectoparasites	Skin lesions
Pseudomonas	0%	10·3%	11·1%	19·5%	12·9%	14·3%	33·9%
Proteus	0	13·6	17·8	22·2	12·9	21·4	41·0
Coliforms	5·7	8·4	22·2	14·8	7·7	21·4	28·8
Haemolytic streptococci	2·9	12·4	22·2	24·2	6·5	21·4	45·8
Other streptococci	30·0	27·7	22·2	20·8	29·0	14·3	23·7
Staphylococci	54·3	70·9	57·8	71·8	80·6	86·0	64·4
Diphtheroids	15·7	12·9	11·1	11·4	19·4	21·4	15·3
Total number of dogs in each category	35	155	45	149	31	14	59

The commonest micro-organisms in both healthy and affected ears were various types of staphylococci, most of which failed to produce pigment on milk-agar medium, irrespective of their coagulase activity. Although 78 per cent. of the strains isolated from otitic material coagulated rabbit plasma slowly, compared with 46 per cent. from healthy ears, there was little evidence to suggest that they were responsible for the onset of otitis and it seemed likely that the presence of numerous potentially pathogenic staphylococci (which was a feature of most acute, uncomplicated cases of external otitis) was due to their ability to penetrate and multiply rapidly within the damaged surface epithelial layers of the initial ear lesion.

On the other hand, although *Proteus* and *Pseudomonas* were not present in healthy external ears, they were almost equally prevalent in the affected ears of dogs which were not also suffering from a skin disease. The importance of pyogenic Gram-negative bacilli in canine otitis was further emphasised by their increased incidence in dogs with a clinical skin complaint, especially those whose ears failed to respond promptly to treatment.

Discussion

It is generally agreed that there is an apparent increased breed susceptibility to canine external otitis which varies, to some extent, with the local breed populations and current changes in fashion. This was well illustrated in the case of Miniature Poodles which rapidly increased in popularity towards the end of the survey and accounted for 18.1 per cent. of all dogs with otitis in 1960, compared with only 3.1 per cent. some three years earlier. On the other hand Spaniels were unaffected by recent trends in fashion and the number suffering from otitis was four times greater than for Collies, which were the most popular of the 32 breeds examined. The effect of local breed popularities on the apparent susceptibilities to otitis was overcome by the breed incidence figures which confirmed that Miniature Poodles, Spaniels and certain other breeds with over-lapping ears were particularly prone to otitis, whereas Collies, Boxers and Greyhounds were seldom affected.

Many clinicians have emphasised the importance of *Otodectes cyanotis* as causal agents of canine external otitis and McGinnis and England (1949) reported that nearly 50 per cent. of their cases were due to ear-mite infestation. Moreover, Jennings (1958) is of the opinion that the presence of even an occasional mite is sufficient to bring about the clinical condition and that in most cases the parasites are acquired from cats. Recently, Frost and Beresford-Jones (1960) found *Otodectes* species in 46 of 100 unselected and untreated cases of external otitis and, although the ears of only 9 dogs were heavily infested while the remaining 37 showed fewer than 3 detectable mites on auroscopic examination, they nevertheless considered that otodectic mange is an important cause of external otitis in the dog.

In this present work ear-mites were seldom numerous and were observed in only 6 per cent. of cases, the majority of which were mild, acute forms of otitis uncomplicated by pyogenic bacteria. Their importance, as causal agents of otitis, is probably limited to mechanical irritation of the external ear which causes the dog to scratch and damage the lining membrane of the meatal canal through which commensal staphylococci and other micro-organisms may enter and multiply.

Of greater significance, as predisposing factors to otitis, was the presence of clinical skin conditions

(*Concluded at foot of page overleaf*)

Current Literature

ABSTRACTS

Physalopteran Infection in an Orangutan. JASKOSKI, B. J. (1960). *J. Amer. vet. med. Ass.* **137.** 307.

Dr. Jaskoski describes a heavy infestation by *Abbreviata caucasica* of an 8 years 9 months old orangutan, found dead in his cage at Lincoln Park Zoological Gardens. According to Dr. Jaskoski, the probable cause of death was heart failure due to gross abnormality of the heart.

Routine faecal examination whilst the orangutan was alive had not revealed the presence of infection.

At necropsy the orangutan's oesophagus was found to be impacted with the nematodes and others were found in the trachea and stomach.

Mönnig describes other physalopteran nematodes as affecting the stomach of wild *Felidae* in China, the East Indies, Africa, and South America. They have been known from the duodenum of a dog, and Mönnig refers to physaloptera occurring in the stomach of the dog and cat in South America. There are further references to a physaloptera species in badgers' stomachs.

Craig and Faust (*Clinical Parasitology*) refer to physalopterasis occasionally in humans.

The life cycle is apparently unknown but arthropods such as beetles and cockroaches may serve as intermediate hosts.

Little is known of the clinical and preventive aspects of physaloptera but Ehlers was successful in expelling the worms from badgers using 0.75 ml. carbon bisulphide orally after fasting. Craig and Faust refer to the administration of carbon tetrachlorethylene as recommended for hookworm.

Little also appears to have been written about clinical symptoms or pathogenesis of physaloptera and with regard to Dr. Jaskoski's report on the orangutan it seems that the parasites were commensalls. Ehler's observations on the affected badgers referred to the animals being thin and quarrelsome and in bad cases the faeces had a tarry appearance. Later they stopped feeding and became paretic. It seems that the parasites occurring in the stomach attach themselves to the gastric mucosa upon which they feed, and they may also suck blood. If they change their site of attachment it seems they leave bleeding wounds which lead on to mucosal erosion.

O. G-J.

Relative Activity of the Broad-Spectrum Antibiotics in Birds as Measured by Clinical Effect. PETERSON, E. H. (1960). *Poult. Sci.* **39.** 960.

Following reports that chlortetracycline was superior to oxytetracycline for the treatment of infectious synovitis in fowls, the author investigated their activity in experimentally induced caecal coccidiosis, intestinal coccidiosis (*Eimeria necatrix* infestation), and fowl cholera (*Pasteurella multocida* infection).

He reports that chlortetracycline showed a clinical advantage over oxytetracycline. Both drugs showed an increased activity when "potentiated" by the administration of terephthalic acid or by reducing the calcium content of the ration or by employing both potentiation methods together. However, the increase in activity shown by chlortetracycline was greater than that shown by oxytetracycline, with the result that the difference between their activity was maintained or widened.

Factors Predisposing to Canine External Otitis.—*Concluded.*

in 38 per cent. of the unselected cases of otitis and in 59 per cent. of the dogs which failed to respond promptly to treatment. Many of the animals so affected were suffering from primary or secondary suppurative otitis or otorrhoea. These findings suggested that the extension of a clinical skin condition to the external acoustic meatus might be responsible for the initial lesion of otitis which, if left untreated, would rapidly become infected with commensal staphylococci or with Gram-negative bacilli, from an external source. Antigenic studies on a large number of strains of *Proteus mirabilis* (to be published) showed that a few chronically affected ears became infected with *Proteus*, during treatment, as a result of auto-infection from the intestines.

Although bacteria and fungi are rarely of aetiological importance in canine external otitis, the presence of secondary infections with pyogenic organisms frequently gives rise to marked tissue changes which increase the severity of the clinical condition and render it less amenable to treatment.

Acknowledgments.—I would like to thank Mr. A. R. Withers, Department of Clinical Veterinary Medicine and Surgery, for placing his Departmental records at my disposal and, with members of his Staff, for help in obtaining material.

I am most grateful to Mr. W. W. Gregor for details of the age, breed and sex of dogs examined in the Clinic and for the interest he has shown in this work.

My thanks are also due to Dr. A. Wilson Taylor for helpful criticism.

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Most of the tests were made against caecal coccidiosis, and the author concedes that the strain of *Eimeria tenella* used may have been more sensitive to chlortetracycline than to oxytetracycline.

The pasteurella, however, showed identical *in vitro* sensitivity to both drugs. The author suggests that in all clinical conditions of birds where systemic activity of the antibiotic is required chlortetracycline will show an advantage over oxytetracycline in availability at the seat of disease when the two drugs are given orally.

A Relationship Between Coccidiosis and Vitamin A Nutrition in Chickens. ERASMUS, J., SCOTT, M. L., & LEVINE, P. P. (1960). *Poult. Sci.* **39.** 565.

The infection of 3-week-old chicks with a mixture of caecal and intestinal coccidiosis (*Eimeria tenella* and *E. acervulina*) was followed by a reduction in the vitamin A level in their livers.

The recovery of infected chicks was improved by feeding additional vitamin A.

Use of Mycostatin in the Drinking Water for the Treatment of Crop Mycosis in Turkeys. WIND, S., & YACOWITZ, H. (1960). *Poult. Sci.* **39.** 904.

Turkey poults whose crops had been experimentally infected with *Candida albicans* were treated with various concentrations of nystatin in the drinking water. Sodium lauryl sulphate was used to aid dispersion of the drug.

Levels of 62.5 to 250 parts per million, given continuously for 5 days, gave effective control of the infection. Water consumption was not reduced and in 2 of the 3 experiments there was a tendency towards improved weight gains E. A. G.

Tail Sores in Pigs. (Translated title). HAGEN, O., & SKULBERG, A. (1960). *Nord. VetMed.* **12.** 1.

The author points out that tail sores lead to pyaemia with lungs in particular being affected, and are indirectly responsible for a considerable proportion of pig carcasses which are condemned. In the period 1/1/59 to 30/6/59, 135 pig carcasses were condemned in Oslo slaughterhouse. Of these, 56 were condemned because of pyaemia and in 43 of these pyaemia cases there were tail sores. The author investigated conditions at a number of the piggeries which had sent in affected pigs. These tended to be large piggeries. In one typical piggery large numbers of piglets about 8 to 10 weeks old were bought in and housed in large batches of about 40. Where tail biting occurred, generally only one or a few piglets were responsible. These should be isolated as soon as suspected. Feeding only purchased concentrates and water is regarded as a contributory factor. The bitten tails should be treated or amputated to avoid pyaemia.

The Hygienic Significance of Latent Salmonella Infections in Slaughtered Animals. (Translated title). SANDBU, P. (1960). *Nord. VetMed.* **12.** 29.

Because of the apparent increase of salmonella infections in man it is important to know to what extent slaughtered animals which have been through an outbreak of the disease are infective and what are the most reliable meat inspection methods for detection. Twenty-three calves from a few days to 3 months old, from a herd which had been infected with *Salm. typhimurium* appeared to be healthy and had negative faeces prior to slaughter although most of them had been infected. With 1 exception the carcasses appeared normal and there were only slight pathological changes in the organs. In 17 of the 23 cases *Salm. typhimurium* was isolated from the mesenteric lymph glands (best site) or from the organs, particularly the liver. In no case was the flesh infected but in 5 cases the surface of the meat gave positive samples, presumably due to contamination from faeces on the skin, from a contaminated knife, or while infected organs were being removed. H. B.

Die paratuberkulöse Enteritis der Schafe. (Paratubercular enteritis in Sheep.) ALMEJEW, C. S. (1959). *Mh. VetMed.* **14.** 429.

Professor Almejew gives a description of the pathohistological findings in 81 sheep. Of these, only 11 had been clinically sick (inappetence, increased thirst, thin brown faeces), the others being detected as possible paratubercular animals following positive reactions to intracutaneous avian tuberculin. The pathological lesions were mainly confined to the ileum, caecum, and colon, the intestinal wall being in parts 3 to 5 times the normal thickness. The mucous membrane was thickly folded longitudinally and transversely. Associated lymph nodes were enlarged and oedematous. The author states the paratubercular changes to be characteristic of a specific inflammation. Histologically, an acute inflammation may be recognised apart from a chronic catarrhal process. In acute cases necrotic and proliferative changes occur in adjoining sections of bowel, the former often being more prominent. The specific feature is tissue hyperplasia, with a predominance of polymorphs present. The early and continued presence of polymorphs in the basal layer of the mucous membrane demonstrates that the reticulo-endothelial system is the origin of the process. (3 photographs.)

Eine aussergewohnliche Herzhemmungsbildung bei einew Schwein (Foramen primum presistens). An unusual cardiac abnormality in a pig. JENTZSCH, K. D. (1959). *Mh. VetMed.* **66.** 400.

A 4½-month-old Sattel pig died after a 2-day illness. *Post-mortem* revealed a persistent foramen in the medial septum between left and right auricles, which was the obvious cause of death, though no circulatory disturbance was noted during life. There are 2 photographs, together with 4 diagrams which illustrate the probable embryological development. This is the first instance of a genuine persistent primary foramen to be reported in any domestic animal in the German literature. A. D. W.

The Victoria Veterinary Benevolent Fund

A meeting of the Council of the Victoria Veterinary Benevolent Fund was held at No. 10 Red Lion Square, London, W.C.1., on October 27th, 1960, when the following members were present:—

Mr. H. W. Dawes (President) in the Chair, Messrs. E. P. Barrett, H. E. Bywater, J. Birrell, E. Clark, Major J. J. Dunlop, Mrs. M. Field, Professor C. Formston, Mrs. D. I. Glover, Mr. J. M. Ingram, Professor J. McCunn, Miss V. Tillemont-Thomason, Capt. Wm. Watt, Mr. E. Wilkinson, and Major W. H. Wortley.

In attendance: Mr. R. V. Thatcher (solicitor) and the secretary, Mr. C. W. Francis.

Minutes

The Minutes of the previous meeting, held on June 28th, 1960, having been published in THE VETERINARY RECORD were taken as read and signed.

Apologies for absence

The secretary reported apologies for absence from the following:—

Mr. G. Atkinson, Professors G. F. Boddie, and T. J. Bosworth, Mr. J. A. Flynn, Dr. R. E. Glover, Mr. G. N. Gould, Professor R. Lovell, Mr. D. H. L. Madden, Professor L. P. Pugh, Mr. A. Spicer, Major C. W. Townsend, Mr. W. J. B. Robson, and Professor W. L. Weipers.

Obituary

The secretary reported the death of Mr. G. H. Locke, M.R.C.V.S., which took place on October 21st. Mr. Locke represented members of the Fund on the Council from 1922 to 1944. The President moved from the Chair that a letter be sent to his widow and family expressing the Council's deepest sympathy in the great loss which they had sustained. This was carried in silence, all the members standing.

Correspondence

(i) (a) *Ladies' Guild.* The secretary read a letter from the treasurer enclosing cheques of £1,000 and £500, which were to be allocated to the Current Relief and the Special Gift accounts, respectively, these sums having been voted to the Fund at the Annual General Meeting of the Guild recently held in Glasgow. After the President and members had referred in warm terms to the great work of the members of the Guild which had resulted in the raising of these magnificent amounts, *it was unanimously resolved* that this meeting places on record its appreciation and grateful thanks to the members of the Ladies' Guild for their splendid efforts over the past year, and for the handsome sum of £1,500 donated to this Fund by the Guild.

(b) *Treasurer, Ladies' Guild.* The meeting, having learned with much regret that Mrs. D. I. Glover had tendered her resignation as treasurer of the Ladies' Guild, an office she had filled ably for the past 17 years, *it was unanimously resolved* that a letter from the President, expressing the Council's appreciation of her valuable services to the Guild, be sent to Mrs. Glover.

(ii) *Major James Harrison's Bequest. Farm land at Ditchling.* A letter dated October 21st, 1960, from Messrs. R. H. & R. W. Clutton, the Council's surveyors, conveying an offer from the tenant's agents for the freehold of this land, was submitted to the meeting.

After much discussion, the solicitor was instructed to write to Messrs. R. H. & R. W. Clutton, expressing the views of the Council as to the further action to be taken in this matter.

(iii) *Nominations to Council.* The secretary submitted the following:—

(a) A letter dated October 6th, 1960, from the hon. secretary, Scottish Metropolitan Division, stating that Professor G. F. Boddie had been appointed to succeed Mr. H. W. Wilson as the Division's representative on the Council.

(b) A letter dated August 25th, 1960, from the hon. secretary, Kenya Veterinary Association, intimating that Mr. J. T. R. Evans had been appointed to represent that Association on the Council.

Summer Gifts

The secretary reported that the executive committee, with the assistance of the treasurer of the Ladies' Guild, agreed on special gifts amounting to £251, being allocated to 32 recipients of the Fund, and that these gifts were duly despatched on June 29th.

It was resolved that the action of the executive committee in sending out gifts in June, 1960, amounting in all to £251, as detailed in the list submitted to the meeting, be, and the same is hereby, approved.

Hunting Trust

The secretary reported that the surplus in the Hunting Fund as at December 31st, 1959, amounted to £1,182 0s. 9d., and that in accordance with the terms of the Trust this sum was transferred by the Trustees to the Victoria Veterinary Benevolent Fund on June 30th last.

Investments

The treasurer reported that the executive officers, after careful consideration, decided to invest the amount received from the Hunting Trust, namely £1,182 0s. 9d., in the purchase of the following securities:—

£1,000	on loan to the Swindon Corporation for a period of fifteen years @ 5¾ per cent.
£182.0.9	in £299.15.11 British Transport 3 per cent. Guaranteed Stock, 1978/88.

It was resolved that the action of the executive committee be approved.

Accountant's Report

The secretary read the following report which was approved, a vote of thanks being accorded to the respective donors:—

Since the previous meeting of Council, the following special subscriptions and donations have been received:—

	£	s.	d.	£	s.	d.
Subscriptions						
V.V.B.F. Ladies' Guild				1,000	0	0
Donations						
British Timken Show	1	1	0			
Cranleigh & W. Surrey Farming Club	2	2	0			
Devizes Gymkhana & Horse Show	2	2	0			
Guildford Horse Show	1	1	0			
Lancashire Veterinary Association	10	0	0			
Lincolnshire & District Division	10	0	0			

Midland Counties Veterinary Association Dance	41	18	0			
Nidderdale Agric. Society Canine Show	1	1	0			
North of Scotland Division ...	10	0	0			
Scottish Metropolitan Division (Special Appeal)	38	17	0			
"In memory of the late Walter Scott" (contributions by fifteen organisations and individuals)	39	7	0			
Serth, G. W.	3	3	0			
Windsor Dog Show	10	10	0			
				171	2	0
Collecting Boxes						
Association of State Veterinary Officers	9	0	0			
British Small Animals Veterinary Association	4	4	0			
Eastern Counties Veterinary Society	8	3	3			
Essex Veterinary Society 1 17 9						
1 11 3	3	9	0			
Holmes, J. W. H.	7	10	0			
Mid-West Division	1	3	8			
North of Scotland Division ...	11	10	0			
Scottish Metropolitan Division ...	7	2	0			
Whicher, A.	3	16	0			
				55	17	11
				£1,226	19	11

Cases

No. 152. Widow aged 83. The secretary reported that the Council's instructions given at the April meeting, for a supply of coal to be delivered in this case over the summer months had now been carried out, and a letter from the recipient expressing grateful thanks was read to the meeting. It was resolved that a similar gift be made during the winter months.

No. 245. Widow aged 75. The President reported correspondence he had received from a local practitioner in connexion with this case. As this recipient was now agreeable to accepting help from the National Assistance Board, the question of adjusting the grant in this case was left in the hands of the executive committee.

No. 266. Daughter of late member, aged 62. In view of a letter received by the secretary from the National Assistance Board on October 10th, it was agreed that the grant in this case be increased to 15s. p.w. as from November 1st.

No. 304. Member, aged 62. Son, aged 32, and two daughters, aged 27 and 16. Unable to practise his profession owing to his health. In view of a report on this case it was resolved that the grant payable to this member be discontinued with the December payment.

No. 324. Widow aged 67. This case having been reviewed, it was agreed that the grant be not disturbed for a further twelve months.

No. 326. Widow aged 33. Three children between the ages of 9 to 2½ years. After careful consideration of this case it was decided to make a grant of £5 p.w. to this applicant as from November 1st.

No. 327. Widow aged 37. Seven children between the ages of 12 to 3. The secretary reported the receipt of a letter respecting the elder son's educational expenses. It was resolved that a grant of £1 p.w. be made in this case.

No. 334. Widow aged 70. In view of a local member's report on this case, it was agreed that as from December 1st, the grant to this recipient be £2 p.w.

No. 335. Widow aged 52. Two sons, 22 (married) and 16. As the younger son had now obtained employment, it was resolved that the grant in this case be discontinued after the December payment.

No. 336. Widow aged 80. The secretary reported that the small repairs to her house, which had been authorised by the Council, had now been carried out. It was resolved that the account be paid by the Fund.

No. 337. Daughter of late member, aged 44. Child aged 5. The executive committee reported on the correspondence which had been received since the previous meeting and the action taken thereon, which was approved.

No. 339. Widow aged 36. Two children, son, aged 3, and daughter (blind) aged 9. A recent letter explaining her present position having been carefully considered, it was decided to continue the grant until the next meeting.

No. 340. Member aged 73. The executive committee reported that since the last meeting it had ascertained from the National Assistance Board the Fund could make a grant of 15s. p.w. which the committee had arranged to be paid to this recipient as from July 1st. It was resolved that the action of the executive committee be approved.

No. 341. Member aged 86. Crippled with arthritis. Wife bedridden and lost her speech and in nursing home. Son (married) an invalid. Daughter, aged 46, helping to support home. Receiving grant of £3 p.w. Executive committee reported that this member died on August 13th and that in the special circumstances, it had decided to continue the grant until this meeting. It was resolved that the action of the executive committee be confirmed; that the grant be continued; and that the executive committee be empowered to take such further action as it deems necessary after the daughter has completed a form of application.

No. 342. Widow aged 47. Two children, ages 15 and 11, both at school. The executive committee reported that the mother had taken a part-time appointment to augment her small income. It was resolved that the action of the executive committee in making a grant of £3 p.w. as from September 1st, be approved, and that the grant be reconsidered at the next meeting.

No. 343. Widow aged 85. Suffers with heart trouble. Has a married daughter, aged 49, in delicate health, living with her. The executive committee stated that having regard to a member of Council's report on his recent visit to this case, it felt this application was one which ought to be dealt with by the Council. It was resolved that a grant of £2 p.w. be made to the widow, to take effect as from November 1st.

No. 344. Widow aged 86. Crippled with arthritis, and now in a nursing home. Has a married daughter and a son, both helping towards charges of home. It was resolved that a grant of £2 p.w. be made in this case.

Eire Cases. A report from the Veterinary Medical Association of Ireland's Scrutinising Committee, dated October 7th, on cases Nos. 252a, 270, 273, 281, 322, 331, was submitted and considered, and the recommendation contained therein that the present grants be continued in all cases, excepting No. 270, which should be discontinued at the end of the year, was adopted.

10. *Change of Address*

The President informed the meeting that as the Royal College of Veterinary Surgeons had recently disposed of its properties in Red Lion Square, and would be leaving at the end of the year, this would probably be the last Council meeting at the present address. This was an approprite occasion for the Council to express its gratitude to the Royal College for the generous accommodation which it had provided for their meetings, etc. at No. 10 Red Lion Square over the past forty-three years.

The President proposed from the Chair the following resolution, which was carried unanimously:—

> That this meeting places on record its grateful thanks to the Council of the Royal College of Veterinary Surgeons for the accommodation which it has generously provided for meetings of the V.V.B.F. Council, etc. at No. 10 Red Lion Square, over the past forty-three years.

11. *Date of Next Meeting*

It was agreed that the date and place of the next meeting of Council be left in the hands of the executive committee.

News and Comment

PERSONAL

Engagements

AXFORD—HEELEY.—The engagement is announced between Roger Francis Ernest Axford, M.A., VET.M.B., M.R.C.V.S., younger son of Mr. and Mrs. E. C. Axford, of Horbury, Yorkshire, and Rachel Mary Heeley, daughter of Mr. and Mrs. A. Heeley, of Woodend Drive, Glasgow.

MATSON—GRAY.—The engagement is announced between Basil Matson, B.V.SC., M.R.C.V.S., younger son of Mr. and Mrs. A. L. Matson, of Umtali, Southern Rhodesia, and Jane Gray, B.SC., only daughter of the late Mr. M. Gray and Mrs. Gray, of Milton Abbot, Devon.

Births

CALDER.—On January 7th, 1961, at St. Mary's Hospital, to Joyce, wife of Ronald H. Calder, M.R.C.V.S., Melton Mowbray, a son, Ian William, brother for Carolyn and Jennifer.

CLARKSON.—On January 12th, 1961, to Kenneth and Marjorie E. Clarkson, M.R.C.V.S. (*née* Millar), of Leatherhead, Surrey, a son.

GREEN.—On December 31st, 1960, to Susan, M.S.R.(R)., wife of Roger Green, B.VET.MED., M.R.C.V.S., of Holsworthy, Devon, a son, Richard Douglas.

NUNNELEY.—On January 14th, 1961, to Laurette, wife of David Nunneley, B.V.SC., M.R.C.V.S., of 32 Greenfield Avenue, Northampton, a daughter, Tessa, sister for Christopher and Jane.

SLATER.—On December 24th, 1960, to Anna, wife of Duthie Slater, M.R.C.V.S., D.T.V.M., Veterinary Research Dept., Lagos, Nigeria, a daughter, Jane Laura.

WHITELEY.—On December 31st, 1960, to Ann (*née* Emmett) wife of Hedley Whiteley, B.V.SC., M.R.C.V.S., 32 Windsor Close, Beverley, E. Yorkshire, a son, Peter Milton, brother for Andrew.

COMING EVENTS

January

25th (Wed.). B.S.A.V.A. METROPOLITAN REGION. Meeting at the Royal Society of Medicine, 1 Wimpole Street, London, W.1, 7 p.m.

STAFFS. AND DERBYS. VETERINARY CLUB. Meeting at the White Hart Hotel, Uttoxeter, 8 p.m.

SOUTH-EAST MIDLANDS VETERINARY ASSOCIATION. General Meeting at The Franklins Gardens Hotel, Northampton, 7.30 p.m.

26th (Thurs.). EASTERN COUNTIES VETERINARY SOCIETY. Special General Meeting in the Lecture Theatre of the School of Veterinary Medicine Madingley Road, Cambridge, 2.30 p.m.

WESTERN COUNTIES DIVISION. Meeting at the Rougemont Hotel, Exeter, 2.30 p.m.

27th (Fri.). NORTHERN IRELAND VETERINARY ASSOCIATION. Meeting at the Whitla Medical Institute, College Square North, Belfast, 7 p.m.

30th (Mon.). EAST YORKSHIRE CLINICAL CLUB. Meeting at the Talbot Hotel, Malton, 8 p.m.

February

1st (Wed.). DUMFRIES AND GALLOWAY DIVISION. Meeting in the Queensberry Arms Hotel, Arran, 2.15 p.m.

WESTERN COUNTIES VETERINARY ASSOCIATION. DEVON BRANCH. Meeting at the Tavistock Golf Club, Tavistock, 7.30 p.m.

B.S.A.V.A. REGION 3. A Meeting at The Canine Health Centre, Animal Health Trust, Kennett, Nr. Newmarket, 2.30 p.m.

2nd (Thurs.). WEST OF SCOTLAND DIVISION—CLINICAL CLUB. Meeting at the University Veterinary School, 83 Buccleuch Street, Glasgow, C.3, 7.30 p.m.

4th (Sat.). B.S.A.V.A. SOUTHERN COUNTIES REGION. Meeting at the Royal Hotel, Winchester, 6.30 p.m.

7th (Tues.). 12th Anniversary Dinner-Dance of the University of Bristol Centaur Society at the Berkeley, Bristol.

8th (Wed.). SUSSEX VETERINARY SOCIETY. Ordinary General Meeting at the Dudley Hotel, Lansdowne Place, Hove, 2.30 p.m.

SCOTTISH METROPOLITAN DIVISION. Annual General Meeting at the Royal (Dick) School of Veterinary Studies, 2.30 p.m.

9th (Thurs.). Annual Ball of the Royal (Dick) School of Veterinary Studies in the Assembly Rooms, George Street, Edinburgh, 9 p.m.

SOCIETY FOR THE STUDY OF ANIMAL BREEDING. 9th Annual General Meeting at the Royal Society of Medicine, 1 Wimpole Street, W.1, 10.30 a.m.

ESSEX VETERINARY SOCIETY. Meeting at the George Hotel, Colchester, 7.30 p.m.

15th (Wed.). SOUTH EASTERN VETERINARY ASSOCIATION. Annual General Meeting at the Royal Star Hotel, Maidstone, 7.30 p.m.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Somerset. Rydings Farm, Wrington, Bristol (Jan. 10).
Stirling. Bogton Farm, Larbert (Jan. 16).
Sussex. Crouches Farm, East Hoathley, Lewes (Jan. 13).
Wigtown. Bathaskine, Glenluce (Jan. 16).

Foot-and-Mouth Disease

Fife. East Bowhill, Cardenden (Jan. 12).
Norfolk. Martins Farm, Hindolveston, Dereham (Jan. 10); Low Farm, Ringland, Norwich (Jan. 11).

Fowl Pest

Berks. 2, Surrey Villas, Hyde End Road, Spencer Wood, Reading (Jan. 10).
Essex. South Leigh, Nevendon Road, Basildon (Jan. 10); Bowling Alley House, Little Baddow, Chelmsford (Jan. 12); Brook Farm, Burnham-on-Crouch; Mansion House

Farm, Altthorne, Chelmsford; 148, Fambridge Road, Maldon (Jan. 13); Sunfield Farm, Highlands Hill, Maylands, Chelmsford (Jan. 14); 139, Fambridge Road, Maldon (Jan. 15); Frith View, Mayland, Chelmsford; No. 1 Council Bungalow, Mayland, Chelmsford (Jan. 16).

Hants. The Clock House Poultry Farm, Curdridge, Southampton (Jan. 13); Madden Farm, Hollywater, Bordon (Jan. 15).

Kent. The Billet House, Crowhurst Lane, Ash, Sevenoaks (Jan. 10).

Lancs. Buckley Hall Farm, Ribchester, Preston (Jan. 12); Ashlea, Shaw Green, Euxton, Chorley (Jan. 14).

Leics. Welby Lodge, Welby, Melton Mowbray (Jan. 12).

Lincs. Shoff Farm, North Drove, Quadring, Spalding (Jan. 10); Aerodrome Farm, Cranwell Village, Sleaford (Jan. 14).

London. 226, Walm Lane, Cricklewood, London, N.W.2 (Jan. 10).

Norfolk. Hawkshill, Great Witchingham, Norwich; Holly Farm, Tivetshall St. Mary, Norwich; Cooks Farm, Wingfield, Diss; The Turkey Farm, Beech Road, Taverham, Norwich; Court House, Rollesby, Great Yarmouth (Jan. 10); Abbey Farm, Aylsham, Norwich; Cannon Field, Brandon Road, Thetford; Grange Farm, Salthouse Road, Rackheath, Norwich (Jan. 11); Jubilee Hall Farm, Cranworth, East Dereham; Pages Farm, Guestwick, Dereham; Rookery Cottage, Stradbroke, Diss; Caister Road Allotments, Group C, Great Yarmouth; Hilltop, Tacclenston, Norwich; Priscilla Close, West Earlham, Norwich (Jan. 12); Rose Cottage, Rickinghall, Diss; Abbey Farm, Hoxne, Diss; Beech Farm, Taverham, Norwich; 1, Abbey Terrace, Hoxne, Diss (Jan. 13); Dairy Farm, Reepham Road, Taverham, Norwich; 86, Fakenham Road, Drayton, Norwich (Jan. 14); North Lane Farm, Stradbroke, Diss (Jan. 15); Meadow View, Thurton, Norwich; Honeysuckle Cottage, Black Street, Winterton, Great Yarmouth (Jan. 16).

Suffolk. South Cove House, Wrentham, Beccles (Jan 10); Wangford, Beccles; Church Farm, Whatfield, Ipswich (Jan. 11); Bell Farm, Carlton Colville, Lowestoft (Jan. 12); Theberton House, Leiston; Little Saxtead Green, Saxtead, Woodbridge (Jan. 13); Mall Green Farm, Copdock, Ipswich (Jan. 14); Wrentham House, Wrentham, Beccles; Home Farm House, Therberton, Leiston (Jan. 16).

Surrey. Sunnyacres, Reigate Road, Hookwood, Horley (Jan. 15).

Sussex. Ashurst, Steyning (Jan. 13).

Swine Fever

Ches. Badgers Bank Farm, Hunsterson, Nantwich (Jan. 13); Crewe Road, Wheelock, Sandbank (Jan. 16).

Flints. Cwybr Ucha Piggeries, Rhyl (Jan. 11).

Montgomery. The Lane, Bettws, Newtown (Jan. 11).

Salop. The Buildings Farm, Baschurch, Shrewsbury (Jan. 16).

Staffs. Gorsby Hill Farm, Marchington Woodlands, Uttoxeter (Jan. 16).

Yorks. North Riding Piggeries, Hutton Hill Farm, Ripon (Jan. 11); Bridge Grange, Bedale; Prospect House, Holme-on-Spalding Moor; Rubbish Tip, South Middle Howe Pit, Couth Cave Road, Walkington (Jan. 12); Old Road, Home-on-Spalding Moor (Jan. 13); 26, Main Street, Scholes, Leeds; Elm Tree Farm, Swainby, Northallerton (Jan. 16).

Letters to the Editor

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

The Economics of Veterinary Services to the Stock Owner

Sir,—I would be glad if you would kindly allow me, through your correspondence columns, to put forward the following points of view.

The study of agricultural economics is recognised as essential and beneficial to the agricultural industry. It seems important, however, that more attention should be paid to a specialised part of it, *viz.* the economics of veterinary services to the stock owner.

From fairly close observation on treated and untreated animals, flocks and herds, I have made some attempt to assess the economic results of veterinary services. Probably it is safe to conclude that the average benefit to the stock owner is five or six times his outlay on these services.

How interesting it would be if study and investigation could be initiated, which would through time enable the veterinarian to understand the types of veterinary service most helpful to the farmer!

During the last war the Survey Committee of the N.V.M.A. made very creditable efforts to assess the loss from various epidemics, and our Animal Health Division is, I understand, carrying out useful surveys of the incidence of disease. Increased whole time study could bring facts to light; at any rate it does seem important that knowledge should be established and kept up to date, on the economics of veterinary services to the stock owner. From this knowledge might come indications as to how veterinary service could be applied in our national economy.

A service "loaded," so to speak, with multiple return for outlay seems to deserve very widespread application.

Yours faithfully,
A. J. KENNEDY.

Thornhill,
Dumfriesshire.
January 17th, 1961.

PS. Since writing the above my attention has been drawn to Professor Beveridge's excellent paper (*Vet. Rec.* **72.** 810). I venture still to submit my letter, which may serve to renew interest in this important subject.

The Use of Promazine Hydrochloride in Equine Practice

Sir,—Since re-reading Mr. Chamberlain's letter in your issue of December 17th, 1960, and my letter in the issue of December 31st, regarding the use of promazine hydrochloride in equine practice, I feel that Mr. Chamberlain and I are expecting more of this drug than we should.

"Sparine" is, after all, a tranquilliser, not a sedative nor an analgesic. I very much doubt what benefit it could give when used prior to any surgical or other painful interference. Even the insertion of a needle for local anaesthesia would cause as much pain as it would if the drug had not been administered.

The use of tranquillisers, I think, should be con-

fined to instances where no painful procedure follows, but where the animal shows nervous excitement, *e.g.* the horse that is difficult to box, or is a bad traveller; the horse that is difficult to clip, or shoe, or dress its teeth, etc.

If we had a tranquilliser that was 100 per cent. efficacious in these cases, I feel it would be a great boon to the profession.

Yours faithfully,
A. H. ROBB.

16 Ward Street,
Glasgow,
C.4.
January 12th, 1961.

The Veterinary Nurse-Receptionist

Sir,—In May, 1957, I wrote a letter to THE VETERINARY RECORD, which was printed under the heading "The Status of the Veterinary Nurse-Receptionist." It concerned the lack of progress being made towards the establishment of a training scheme for lay assistants like myself. Nearly four years later, I am still wondering how long it takes members of your profession to "actively consider" a laudable suggestion.

I have also grown weary of waiting for someone to recognise my existence by giving my status a suitable label. If the eleven years I spent accumulating knowledge and experience in small-animal practice are considered so worthless, it is not surprising that I have decided to leave this soul-destroying occupation to others less conscientious than myself.

At the risk of sounding presumptuous, may I point out that animals' lives can quite often depend on the speed and efficiency of an unqualified assistant, especially during a major surgical operation. (What if she drops the last available pair of forceps at a crucial moment, or perhaps cannot lay her hands on the coramine in an emergency?)

A qualified assistant is a veterinary surgeon, so why not call him one? The title of Veterinary Assistant could then be used more aptly to identify other *trained* staff.

Yours faithfully,
VALERIE K. JOHNSTON.

Hazelhurst,
Withbean Road,
Brighton, 5.
January 12th, 1960.

Human Brucellosis Risks from Swine

Sir,—The symposium on brucellosis in THE VETERINARY RECORD of October 29th, 1960, is the most comprehensive and authoritative résumé of this disease in cattle and man that I have been privileged to read.

It rather surprises me, however, that no mention was made of human brucellosis risks from swine. In some sections of the United States, especially in the midwest, the number of human cases due to *Brucella suis* exceeds those caused by milk-origin *Brucella abortus*. In one village where the disease assumed endemic status, the raw milk of a single dairy was contaminated by the *suis* strain. In Iowa, the *melitensis* strain has also been isolated from naturally-occurring field infection in swine. Industrial insurance records of midwest abattoirs show that most cases are among pork-workers.

Unfortunately, *Brucella suis* is the most malignant human pathogen, for the porcine strain shows a distinct predilection toward osteomyelitis and spondylitis.

As a zoonosis, we might well keep in mind that brucellosis is a disease of mammalia and not of dairy cows alone.

Yours faithfully,
A. H. QUIN.
Vice President.

Jensen-Salsbery Laboratories, Inc.,
21st and Penn Streets,
Kansas City, 41,
Missouri,
U.S.A.
December 29th, 1960.

Two Unusual Cases

Sir,—I should like to record the following two cases, both seen in general practice in the space of 2 weeks.

A 12-year-old Miniature Poodle bitch, with a history of excessive thirst and urination, breathlessness and a foul smelling breath. There was no cough, diarrhoea or vomiting and the appetite was normal. The heat periods had been irregular, the last being 4 months previously.

At examination the bitch was bright, temperature normal. There was gross tartar and pyorrhoea and halitosis. Tonsils and lymph nodes were normal. Slight systolic murmur; broncho-vesicular breathing; percussion normal. Abdominal palpation revealed no abnormality. Alopecia in sacral region. Urine examination showed no protein or casts.

Conservative treatment was only partially successful, and a laparotomy was advised. On exploring the abdomen, a large ovoid mass was found at the anterior pole of the right kidney, about 2 inches in length and 1 inch in diameter, which had enveloped the vena cava. Euthanasia was carried out.

At *post-mortem* examination there were no macroscopic secondaries. The uterus and kidneys appeared normal, but the ovaries contained small cysts. The tumour was identified as an adrenal cortical adenoma. From the signs produced this was very evidently actively secreting hormones, affecting reproduction, water-salt balance, and hair growth.

The second case was that of a 7-year-old Dalmatian dog with a history of a previous illness some 6 weeks before. This was apparently of a respiratory nature and responded to chloramphenicol therapy. However, from that time the dog had commenced vomiting, nearly always first thing in the morning, the vomit being fluid in nature. The dog was otherwise normal, with a good appetite.

At examination no abnormality could be detected. The urine contained a fair amount of protein, but there were no casts, only sperm and a few epithelial cells.

Gastric sedatives and antibiotics were prescribed without effect, and laparotomy was advised.

On exploring the abdominal cavity, a neoplasm was found in the lesser curvature of the stomach. Gastrectomy was considered, but the client preferred euthanasia.

At *post-mortem* examination the neoplasm was found to be 1½ inches in diameter, on the lesser curvature, and the mucosal surface was ulcerated. The ulcer was about 2 inches in diameter with overhanging edges. The tumour was identified as a lymphosarcoma. There were 2 very large fleshy lymph nodes near the tail of the pancreas. Both kidneys were fibrotic, showed evidence of chronic nephritis, and contained several renal calculi up to 3 mm. in diameter. There were no macroscopic secondaries elsewhere.

It is interesting to note that the dog was bright and showed no signs of urinary disturbance. The only sign was "morning sickness."

The occurrence of the two neoplasms is rare. Cotchin (1959) described 4,187 neoplasms, 9 of which were gastric and 7 adrenal.

My thanks are due to Messrs R. S. F. Campbell and H. B. McCusker, Glasgow University Veterinary School for histological examinations.

Yours faithfully,
H. C. WILSON.

134 Bonnygate,
Cupar-Fife.
January 13th, 1961.

Reference

COTCHIN, E. (1959). *Vet. Rec.* **71**. 1,040.

Umbrellas : Sheep, for the Use of

Sir,—May I seek the courtesy of your columns to warn my colleagues against a difficulty in which I recently found myself?

A client had asked me the best way to immunise his sheep against clostridial infection. As I had thrown all relevant literature in the waste-paper basket I wrote to one of the leading firms for advice. Their reply was most prompt and to the point. They sent me a beautiful picture of Lord Nelson standing underneath an umbrella! I was delighted. There were seven hundred and fifty sheep concerned and I had a little difficulty in persuading my client to buy 750 umbrellas. He is an experienced farmer, and had found that sheep never remember to take their umbrellas if it looks like rain. However, he relented and I started enquiries about Lord Nelson hats to fit sheep. Then I made a horrid discovery. I had been gulled by a lot of sales talk, and the umbrellas had nothing to do with the treatment at all.

May I therefore suggest to my colleagues, that they continue to throw this kind of literature in the waste-paper basket, and to rely upon those firms who understand the fact that most veterinary surgeons are able to read.

Yours faithfully,
A. C. DIXON.

254, High Street,
Berkhamsted.
January 12th, 1961.

Foot-and-Mouth Disease

Sir,—Your reports on the foot-and-mouth disease outbreaks in Great Britain indicate a very sorry state of affairs.

The agricultural community has for some time past been in favour of halting the flow of Argentine diseased beef into the United Kingdom, and in February, 1958, representations to this effect were made to the Ministry.

On the other hand, H.M. Government has shown no intention of stopping this traffic (even though, in their own words, 62 primary outbreaks have had their origin therein, in the 5 years up to 1959), while the British veterinary profession adopts an entirely neutral attitude.

There is, however, one possible solution which may satisfy both parties to the dispute.

There was published in THE RECORD some years ago an article by a veterinary research worker which showed that the virus remains viable in the bone marrow and offal, but not in the carcase meat.

I would suggest, therefore, that only boneless carcase meat be permitted to enter the United Kingdom from South America, all other forms of meat, including offal, being entirely excluded.

I might add that if my advice on this subject, as published in your columns during 1958, had been followed, this disastrous outbreak of foot-and-mouth disease would not have occurred.

Yours faithfully,
J. N. SIMPSON-WHITE.

10504 Walnut Avenue,
South Gate,
California,
U.S.A.
January 8th, 1961.

An Unusual Case of Diabetes Mellitus in a Bitch

Sir,—I was very interested in Mr. Ross's letter of January 14th which describes a case of diabetes mellitus in a bitch, particularly in his reference to the evaluation of urinary diastase (amylase), and the interpretation he places on his findings.

In human work this test for amylase is normally carried out on a sample of urine taken from a mixture of all urine passed during a given 24-hour period. Interpretation of this test is difficult since the clinician is faced with certain variables, *e.g.* the concentration and volume of output of urine. Since this test merely reflects the amylase level of the blood, it is usually considered better to evaluate plasma amylase. This is a very simple test and is accurate.

The plasma amylase test is useful in human medicine even in cases of total obstruction of the ducts, that is where no pancreatic juice is able to enter the small intestine. Abnormal values of amylase are always higher than normal, and I am reliably informed that there is no known condition in the human subject where low readings are of significance. One condition is said to exist where high plasma amylase readings are accompanied by very low urinary amylase values, but this is thought to be a failure in kidney filtration.

I was surprised to learn that this urine test can be helpful as an indication of pancreatic function.

since it was not devised as such nor normally used for this purpose. It is a bit wild to claim that this test pointed to a low production of pancreatic amylase when it was clear enough that production of trypsinogen and lipase was not greatly impaired.

It is quite naughty to interpret a test, carried out before death of the patient and presumably as an aid to diagnosis, only after the *post-mortem* examination is completed. This amounts to "unfair tactics," for biochemical tests as ancillary aids to diagnosis are utterly useless unless they can be interpreted with accuracy during the patient's life. All clinicians can be clever after the event.

Perhaps we shall learn if the writer would have put forward his interpretation of the urinary diastase test in this instance if the dog had still been alive.

Yours faithfully,
GEORGE W. CRIGHTON.

132 Lichfield Road,
Stafford.
January 15th, 1961.

The Future of Animal Husbandry in Overseas Territories

Sir,—Your contributor, Mr. Miller, is to be congratulated on opening for discussion this topic of such moment: animal husbandry, particularly in Africa, is passing through a hazardous phase and the future is far from clear.

It has been my privilege in the past few years to engage on a variety of lesser investigation and research problems in Africa during the course of which I have met many of our colleagues and visited almost all of the veterinary institutions, research and diagnostic laboratories in East, Central and South Africa. Everywhere there are fascinating problems begging for solutions and the need for veterinary surgeons has never been greater.

However, the recent and rapid political developments have precipitated a crisis which in many areas is threatening the future of animal production. For instance, at one laboratory the entire establishment of three veterinary research officers resigned, another was reduced from half a dozen to a single officer, while elsewhere there are almost more American visitors than permanent personnel. In one of the few laboratories fully staffed it was clear that several of the veterinarians were intending only to complete the tour. Furthermore, it was unusual to meet persons who had spent more than a dozen years on research in Africa, and viewing the position as a whole one found it difficult to escape the suggestion, allowing for certain outstanding exceptions, that the profession there was rapidly coming to a standstill.

The problems of our colleagues in that vast continent may seem a little remote to the average practitioner here at home, but certainly this is not true for those who are responsible for the training of veterinary surgeons. In East, West, and Central Africa north of the Zambezi, almost the entire present veterinary surgeon strength emanates from the veterinary faculties of Britain. As a proportion of our profession at home and abroad their numbers are small, but viewed in relation to animal husbandry and industry in Africa their presence determines the very existence of veterinary science. (To the south of the Zambezi in South Africa and Southern Rhodesia a similar statement, to a lesser extent, would also be true, though here the life blood is injected principally by the veterinary faculty at Onderstepoort.)

Thus the future in the overseas territories is almost entirely our responsibility and one wonders just where help can best be given. Perhaps it would be considerably to the benefit of the profession abroad, the countries they serve, and to the advancement of education in Britain if our veterinary schools were to take a greater interest in tropical medicine, for example by liaison with laboratories in Africa with a view to adopting the smaller research and diagnostic centres as field stations of respective veterinary faculties.

Such an association, though at first glance apparently costly and beyond the limited means of university finance, would, I am sure, considerably stabilise the present widespread lack of continuity of research effort particularly in the rapidly emerging territories. At the same time it would provide material, specimens and experience so necessary for the teaching of tropical medicine and so difficult to come by within Britain. What is of even greater importance, it might lead to an every-day contact with the research problems of Africa, many of which can be tackled only in the better equipped and staffed research centres in Britain.

This suggestion is by no means new, and in the medical field outstanding examples of the success of this co-operation are to be seen in such institutes as the London and the Liverpool Schools of Tropical Medicine. Their activities include frequent visits to tropical areas resulting in achievements so outstanding that they surely indicate the part we veterinarians must play.

Yours faithfully,
B. A. MATSON.

London House,
Guildford Street,
London, W.C.1.
January 15th, 1961.

The New Year Honours

Sir,—It might interest her many friends in the profession to be reminded that Mrs. Harold Beeley, whose husband has been proposed as the British ambassador to Cairo, qualified as Miss P. K. Shields.

Mr. Beeley was made a knight commander of the order of St. Michael and St. George in the New Year Honours List and will receive his knighthood at the coming investiture.

Mrs. Beeley was with me as assistant for some years and has carried on a certain amount of veterinary work since going to America.

Yours faithfully,
E. J. HEATHER.

35 Iffley Road,
Oxford.
January 16th, 1961.